A STUDY ON ANALYSIS OF CAUSES , COURSES, AND OUTCOME OF ACUTE RENAL FAILURE IN PREGNANCY

Dissertation Submitted to

THE TAMILNADU Dr.M.G.R. MEDICAL UNIVERSITY

CHENNAI - 600 032

with partial fulfillment of the regulations

for the award of the degree of

MS OBSTETRICS AND GYNAECOLOGY

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY

COIMBATORE MEDICAL COLLEGE AND HOSPITAL,

COIMBATORE

APRIL 2016

CERTIFICATE

This is to certify that the dissertation entailed **"A STUDY OF ANALYSIS OF THE CAUSES, COURSE AND OUTCOME OF ACUTE RENAL FAILURE IN PREGNANCY"** is a bonafide and genuine research work carried out by **DR. K.NEERAJA** in partial fulfillment of the requirement for the degree of Master of Surgery in the Department of Obstetrics and Gynaecology.

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Dissertation Topic : ANALYSIS OF CAUSES, COURSE AND OUTCOME OF ACUTE RENAL FAILURE IN OBSTETRICS.

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Page count:	81
Word count:	7,551
Character count:	42,549
Submission date:	20-Sep-2015 12:29PM
Submission ID:	570759828

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DECLARATION

I hereby declare that this dissertation entitled **"A STUDY OF ANALYSIS OF THE CAUSES, COURSES AND OUTCOME OF ACUTE RENAL FAILURE IN PREGNANCY"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. R. MANONMANI MD., DGO.,** Associate Professor, Department of Obstetrics and Gynaecology, Coimbatore Medical College and Hospital, Coimbatore.

Date : **Dr.K.NEERAJA**

Place :

ACKNOWLEDGEMENT

I express my deep sense of gratitude and heartfelt thanks to **Dr.R.MANONMANI,MD., DGO.,** Associate Professor ,Department of Obstetrics and Gynaecology, Coimbatore Medical College and Hospital, Coimbatore, for her invaluable guidance, constant encouragement, immense patience and great care towards this dissertation.

I am also directly indebted to **Dr.P.SUNDARI, MD.,DGO.,** Professor and Head of the Department, Department of Obstetrics and Gynaecology, Coimbatore Medical College and Hospital, Coimbatore for her profound enthusiasm and keen supervision of this work.

I wish to express my heartful gratitude to **Dr.VATSALADEVI, MD., DGO., Dr.RAJA LAKSHMI, MD.,DGO., DR.PREMAKUMARI, MD.,OG.,** Professors, Department of Obstetrics and Gynaecology, Coimbatore Medical College and Hospital , Coimbatore whose knowledge and experience have guided and inculcated in me a sense of confidence.

My heartful thanks to **Dr. PRABHAKARAN.A, MD., DM.,** Professor, Department of Nephrology, Coimbatore Medical College and Hospital, Coimbatore for his support and contribution for this study.

My heartfelt thanks to my Assistant Professors **Dr.V.NANDHINI, DGO., DNB., Dr. M. SAVITHIRI, DGO., Dr. P. THILAGAVATHY, MD.,OG., Dr.V.GEETHA, M.D.,O.G., Dr.KURINJIPRIYA, M.S.,O.G., Dr. SUMITHRA, M.S.O.G., and Dr.A.DEVILAKSHMI, M.S.,O.G.,** for their kind words and encouragement.

I extend my thanks to **Dr. EDWIN JOE, MD., BL.,** Dean, Coimbatore Medical College and Hospital, Coimbatore.

I am ever grateful to my husband and my father for being a constant source of support and encouragement.

I extend my thanks to my entire Postgraduate colleagues for their support and help.

I thank my Statistician, for his suggestions and for providing a scientific meaning to this study.

Last but not least I would like to thank all my patients without whose cooperation this dissertation would never have seen the light of the day.

Dr.K.NEERAJA

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ABBREVIATIONS

AKI	-	Acute Kidney Injury
ARF	-	Acute Renal Failure
ATN	-	Acute Tubular Necrosis
HELLP	-	Hemolysis, Elevated Liver Enzymes, Low Platelets
AFLP	-	Acute Fatty Liver of Pregnancy
ADH	-	Anti Diuretic Hormone
GFR	-	Glomerular Filtration Rate
PPH	-	Post Partum Haemorrhage
WHO	-	World Health Organisation
DIVC	-	Disseminated Intra Vascular Coagulation
IUGR	-	Intra Uterine Growth Restriction
AST	-	Aspartate Transaminase
LDH	-	Lactate Dehydrogenase
HUS	-	Hemolytic Ureamic Syndrome
TTP	-	Thrombotic Thrombocytopenic Purpura
UTI	-	Urinary Tract Infection
ARDS	-	Acute Respiratory Distress Syndrome
LCHAD	-	Long Chain 3- Hydroxy Acyl – COA Dehydrogenase
WBC	-	White Blood Cell
RBC	-	Red Blood Cell
INR	-	International Normalised Ratio

HIV	-	Human Immuno deficiency Virus
CD 95	-	Cluster of Differentiation 95
LSCS	-	Lower Segment Caesarean Section
NICU	-	Neonatal Intensive Care Unit
CVT	-	Cerebral Venous Thrombosis
BMI	-	Body Mass Index
VBAC	-	Vaginal Birth After Caesarean Section
MTP	-	Medical Termination of Pregnancy
SLE	-	Systemic Lupus Erythematosus
TB	-	Tuberculosis
G	-	Gravidity
P	-	Parity

INTRODUCTION

Acute kidney injury (AKI) formerly called as acute renal failure(ARF) is a most challenging and important complication in pregnancy. It is characterized by sudden loss of renal function along with rise in creatinine levels. It corresponds to nearly 6% to 30% of maternal mortality . Recovery is the rule when it is diagnosed and treated early.

The causes may be

Pre Renal

- Hemorrhage
- Hyperemesis gravidarum
- Congestive heart failure
- Sepsis

Renal

- Acute tubular necrosis(ATN)
- Pyelonephritis
- Renal cortical necrosis
- Thrombotic micro angiopathy
- Pre eclampsia
- HELLP Syndrome

- Acute fatty liver of pregnancy(AFLP)
- Glomerulonephritis
- Medications

Post Renal

- Obstruction

In developing countries like India , AKI remains a frequent and grave complication of pregnancy associated with maternal and fetal mortality. There has been decline in the incidence due to improvement in the antenatal care , early diagnosis and also of the legalization of abortion.

The most common cause of death is due to renal failure from hypertensive disorders of pregnancy and its complications. Most common associated comorbid condition is anaemia.

The aim of this study is to analyse the various causes of acute kidney injury in pregnancy, the factors affecting its course and to determine the outcome of pregnancy among the pregnant women treated at Coimbatore Medical College and Hospital, Coimbatore.

AIMS AND OBJECTIVES

- To study the incidence of acute renal failure in relation to age , parity and duration of pregnancy.
- To analyse the clinical spectrum of presentation in pregnancy.
- To assess the severity of maternal outcome in terms of maternal morbidity and mortality.
- To assess the outcome of pregnancy with renal failure in terms of fetal morbitidity and mortality.

ANATOMY OF KIDNEY

The Kidneys are bean shaped, paired retroperitoneal structures located between the transverse processes of T12 to L3 vertebrae. It measures approximately 11-14 cm in length , 6 cm in width and 4 cm thick. It weighs about 150 gm in adult males and 135 gm in adult females. The left kidney lies superior in position when compared to the right.

Superiorly Adrenal glands lie on the upper pole of kidney.

In the right side , the second part of the duodenum forms the medial aspect of the kidney.

In the left side , the greater curvature of the stomach forms the superomedial aspect.

The tail of the Pancreas overlies the Hilum of the left kidney.

Spleen is located anterior to the upper pole of the left kidney and is attached by splenorenal ligament.

Inferiorly , colon lies on both sides.

Posteriorly, the diaphragm covers the upper third of both the kidneys and the 12 th rib crosses the upper pole of both the kidneys.

The kidneys lies over the Psoas and the Quadratus lumborum muscles laterally.

The two layers of fat (perirenal and pararenal fat) and the renal fascia cover the kidneys.

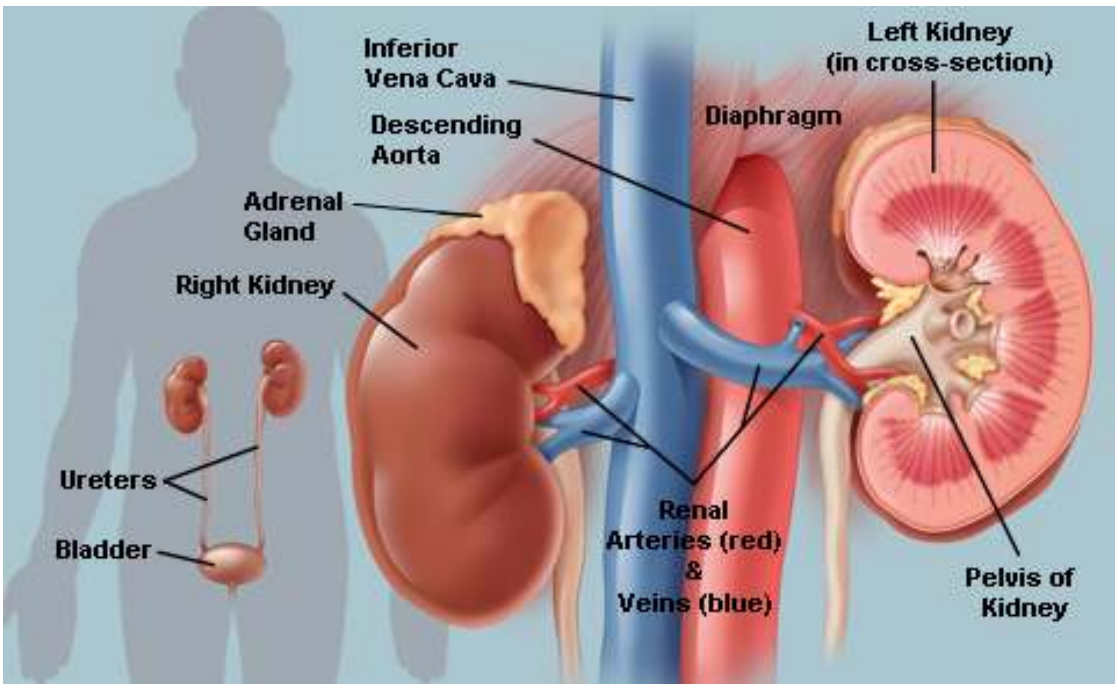


FIG NO 1 ANATOMY OF KIDNEYS

BLOOD SUPPLY

The kidneys receive about 20% of the cardiac output. The main source of blood supply is through paired renal arteries at the level of L2. Renal artery is a branch from the Aorta just before the origin of the Superior Mesentric Artery. It enters through the Hilum of the kidney with the renal vein anteriorly and the renal pelvis posteriorly.

The right renal artery is longer and higher than the left one. Left renal artery is crossed anteriorly by the Inferior Mesentric Vein. Subdivisions of renal arteries are segmental, lobar, inter lobar, arcuate , inter lobular and afferent and efferent glomerular arterioles.

VENOUS DRAINAGE

Fine radicles from the venous ends of peritubular plexuses join to form inter lobular veins which in turn form several stellate vein and end in arcuate veins. Arcuate veins drain it to inter lobar veins which anastomose to form renal vein. Renal vein lies anterior to the renal artery. Left renal vein is longer than the right one. Left renal vein receives the left gonadal vein and drains in to

Inferior Vena cava. Right renal vein is short and drains to Inferior vena cava.

INNERVATION

Both the kidneys are supplied by renal plexus which course along the renal arteries. Input from the sympathetic nervous system triggers vasoconstriction in the kidney thereby reducing renal blood flow.

MICROANATOMY

The kidney is divided into the cortex and medulla.

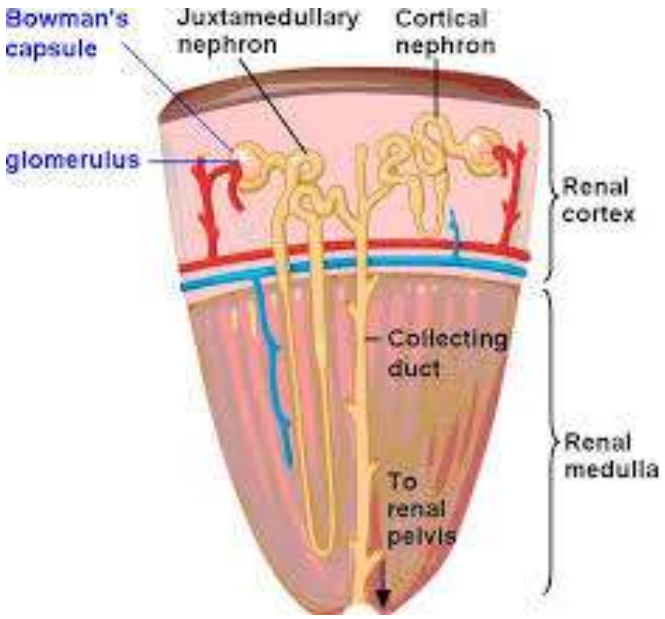


FIG NO.2 MICRO ANATOMY OF KIDNEY

The outer layer is the Cortex that contains:

1. Glomeruli
2. Proximal Tubules
3. Cortical Portions of Loops of Henle
4. Distal Tubules
5. Cortical Collecting Ducts .

The inner layer is the Medulla which is comprised of Renal Pyramids.

The pyramids has the following structures

1. Medullary portions of Loops of Henle
2. Medullary Portions of Collecting Ducts .

Multiple pyramids taper and join forming a minor calyx. Many combine to form a major calyx. The major calyces join and enter a funnel shaped renal pelvis that directs urine into the ureter.

Components of the Nephron:

Approximately one million nephrons comprise each kidney.

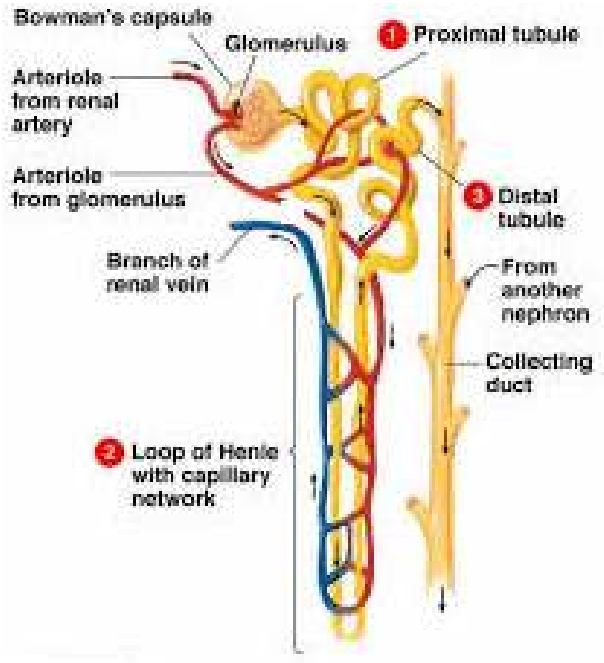


FIG NO 3 STRUCTURE OF NEPRON

The nephron is comprised of following structures

1. Glomerulus
2. Bowman Capsule
3. Proximal Convoluted Tubule
4. Loop of Henle
5. Distal Convoluted Tubule
6. Collecting Duct .

Nephrons are of two types namely

1. Cortical Nephrons
2. Juxtamedullary Nephrons .

Cortical Nephrons constitute about approximately 85 % of the total. They perform excretory and regulatory functions. The remaining 15% is juxtamedullary nephrons which are responsible for concentration and dilution of urine .

Urine Formation

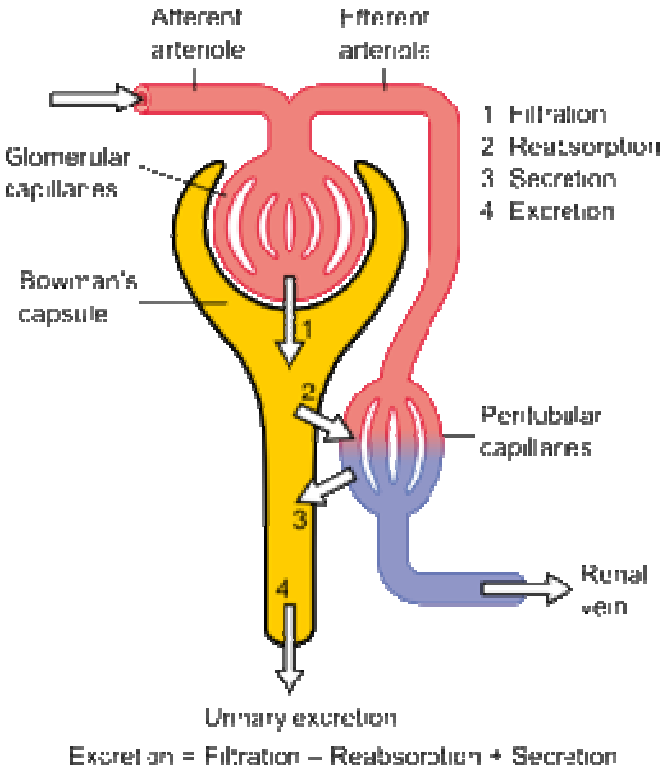


FIG NO 4 URINE FORMATION

Three processes required for urine formation include:

- Glomerular Filtration
- Tubular Reabsorption
- Tubular Secretion .

Glomerulus filters fluid and solutes from blood Proximal Convoluted Tubule and reabsorbs Na+, K+, Cl-, HCO3-, urea, glucose & amino acids. Then the Filtrate moves to the Loop of Henle where Na+, K+ & Cl- are reabsorbed. It also blocks reabsorption of H2O. This dilutes the Urine. Then in the Distal Tubule Na+, K+, Ca++, PO4 selectively reabsorbed . H2O is reabsorbed in presence of Antidiuretic Hormone (ADH). In the Collecting Duct , reabsorption is similar to distal tubule. HCO3- & H- reabsorbed/secreted to acidify urine .

Then the Filtrate leaves hyperosmotic/hypoosmotic depending on the body's requirements .

COMPOSITION OF URINE:

Urine is composed of the following

1. H2O
2. Electrolytes- Na+, K+, Cl-, HCO3-
3. End products of protein metabolism- urea, creatinine, PO4, SO4
4. End products of nucleic acid metabolism- uric acid
5. Breakdown products of phosphoric and sulphuric acid
6. H+ ions excreted bound to buffers such as PO4 and NH3 .

FUNCTIONS OF THE KIDNEY

1. Removal of metabolic wastes .
2. Maintaining fluid and electrolyte balance.
3. Control of Blood Pressure .
4. Metabolism of bone.
5. Maintaining Acid- Base Balance.

CREATININE METABOLISM

Serum creatinine is an important indicator of renal function because it is excreted unchanged by the kidneys. It is synthesized in the liver from the methylation of glycocyamine (guanidinoacetate synthesized in the kidney from the amino acids arginine and glycine) by S-adenosyl methionine. It is then transported to the other organs like muscle and brain through blood where phosphorylation occurs. Phosphocreatine is produced. During this reaction , creatine and phosphocreatine is catalysed by creatine kinase to form creatinine.

Excretion is mainly by glomerular filtration and little by proximal tubular secretion. Hence if there is any defect in the glomerular filtration , creatinine level raises. It also indicates damage to the functioning nephrons.

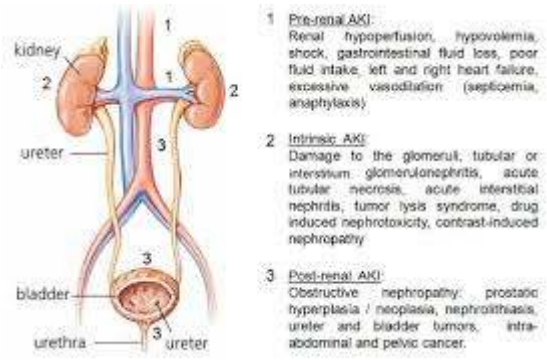


FIG NO 5 TYPES OF ACUTE KIDNEY INJURY(AKI)

PRERENAL ACUTE KIDNEY INJURY

This is due to renal hypoperfusion, hypovolemia, shock, gastro intestinal fluid loss, poor intake of fluids, left and right heart failure, excess vasodilatation as in sepsis and anaphylaxis.

RENAL ACUTE KIDNEY INJURY

This occurs due to damage to the glomeruli,tubules or interstitium, glomerulonephritis, acute tubular necrosis, acute interstitial nephritis or drug induced.

POST RENAL ACUTE KIDNEY INJURY

This may be due to moderate to severe dilatation of the collecting systems due to the causes like gravid uterus, polyhydramnios, kidney stones and enlarged uterine masses like fibroids.

REVIEW OF LITERATURE

ANATOMICAL CHANGES DURING PREGANCY

Increase in the size of the kidneys by about 1 to 1.5 cm due to renal vascular and interstitial space volume expansion.

Physiological hydronephrosis in pregnancy is noted due to dilatation of the calyces , renal pelvis and ureters.

Persistence of this dilatation will be present till the 16^{th} post partum week . This causes urinary stasis and also urinary tract infections.

Mechanisms of the above are due to the following

1. direct action of estrogen and progesterone
2. inhibition of the ureteral peristalsis by prostaglandins
3. mechanical obstruction by the gravid uterus

PHYSIOLOGICAL CHANGES DURING PREGNANCY

Glomerular filtration increases after conception and reaches above 50% in the second trimester and falls to about 20% in the last trimester.

Renal plasma flow also increases which is due to the increase in the cardiac output and inceased renal vasodilatation of afferent and efferent arterioles. This is mediated by nitric oxide ,relaxin and resistance to the action of angiotensin 2. This leads to fall in the blood pressure .

Hence there is increasing heart rate and renin angiotensin aldosterone axis is activated leading to increase in the blood volume and sodium retention.

High GFR will cause increased urate clearance resulting in reduced uric acid levels in pregnancy. Glucose is filtered increasingly which leads to renal glycosuria which is normal in pregnancy.

Increased ventilation in pregnancy will cause chronic respiratory alkalosis.

BIOCHEMICAL CHANGES DURING PREGNANCY

Serum creatinine falls to about 0.5 mg/dl due to increased GFR

Serum uric acid also falls to about 2-3 mg /dl.

Blood urea nitrogen also falls to about 8 – 10 mg/dl.

There will be mild hypo natremia .

Serum bicarbonate is decreased by about 4-5 meq /l . P Co2is decreased by 20 mm HG.

Serum osmolality decreases by about 10 mosm/l during normal pregnancy.

Increased vasopressin produced by the placenta may sometimes result in transient diabetes insipidus during pregnancy.

Proteinuria in pregnancy is defined as more than 300 mg /day of protein excretionwhen compared to 150 mg/dl in non pregnant females. This is due to significant hyperfiltration and reduced tubular reabsorption in pregnancy.

Changes during pregnancy

S.NO		Non Pregnant	Pregnant
1	HAEMATOCRIT	41	33
2	PLASMA CREATININE	1.3 mg/dl	<1mg/dl
3	PLASMA UREA	4-11 mg/dl	3.2-4.4 mg/dl
4	PLASMA ALBUMIN	3.5-4.5 g/dl	2.5-3.5 g/dl
5	PLASMA URIC ACID	4 μmol/l	3.2μmol/l
6	PLASMA BICARBONATE	22-28 mmol/l	18-20 mmol/l
7	URINARY PROTEIN EXCRETION	<150 mg/d	<300 mg/d

ACUTE KIDNEY INJURY

Acute kidney injury is a clinical condition characterized by the decline in the renal function which results in accumulation of nitrogenous wastes . This is a rare but life threatening condition. The two important features of acute kidney injury are rising serum creatinine more than 0.5 mg /dl and oliguria less than 400 ml of urine in 24 hours.

The various causes of acute renal failure in pregnancy are as follows

1. Haemorrhage
2. Hyperemesis gravidarum
3. Hypertensive disorders of pregnancy
4. Abruptio placenta
5. Acute fatty liver of pregnancy
6. Amniotic fluid embolism
7. Systemic diseases like Lupus and vasculitis
8. Incompatible blood transfusion
9. Urinary tract obstruction
10. Sepsis
11. Idiopathic

HAEMORRHAGE

The haemorrhage during pregnancy can occur during both antenatal as well as postnatal period. During antenatal period ,the causes are abruptio placenta and placenta preavia. Severe post partum haemorrhage following delivery can also lead to acute renal failure if correction of blood loss is not done appropriately.

Obstetric haemorrhage constitutes about 24% of the maternal mortality world wide. Post partum haemorrhage is the most common type. Post partum haemorrhage is defined as loss of blood of more than 500 ml within 24 hours of normal delivery and >1000ml in LSCS. It is of two types namely primary and secondary.

Causes of primary post partum haemorrhage

1. Atonic uterus
2. Retained tissues like adherent placenta, retained products of conception
3. Trauma like lacerations,uterine rupture,uterine inversion
4. Coagulation defects

Causes of secondary post partum haemorrhage

1. Subinvolution
2. Placenta accreta
3. Retained products of conception
4. Infection

PPH is an unpredictable event and hence risk factors to be identified and managed correctly . Prevention of PPH is done as per recommended by WHO.

Active management of third stage of labour which includes administration of uterotonics oxytocin 10 units soon after the delivery of the baby, controlled cord traction and uterine massage.

MANAGEMENT

Management involves the following steps

1. Resuscitation
2. Arresting the bleeding either by medical or surgical methods

   Medical-oxytocin, prostaglandins, ergometrine

   Surgical-tamponade methods and uterine compression surgeries

Complications may vary from mild anaemia to severe life threatening like hypovolemic shock, renal failure, disseminated intra vascular coagulation, hepatic failure, adult respiratory distress syndrome and even maternal death. If the blood loss is severe and there is hemo dynamic instability with persistant hypotension, renal filtration is ceased and hence leading to ischaemia followed by acute tubular necrosis. This will result in acute renal shut down and anuria . Late complications like sheehan's syndrome can occur.

Early and adequate replacement of blood products and identification and correction of cause for PPH will help in reducing the various complications.

HYPEREMESIS GRAVIDARUM

It is the condition charaterised by persistant vomiting resulting in severe dehydration, weight loss greater than 3% , electrolyte disturbances and ketonuria which needs hospitalization. It contributes to about 0.3 -1.5% of all pregnancies.

Etiology is hormonal, immunological and psychological factors. It is associated with multiple pregnancy , molar pregnancy and hyperthyroidism.

Raised haematocrit and urea, altered liver function, hypokalemia ,hypochloremic metabolic acidosis and abnormal thyroid function tests are seen.

Complications include electrolyte imbalance, liver dysfunction ,jaundice, acute renal failure, ulcers,esophageal rupture, pnemo thorax, wernicke' s encephalopathy, korsakoff's psychosis and depression.

Intravenous hydration , vitamin supplementation, steroids and ondansetron are effective in treatment.

ABRUPTIO PLACENTA

Placental abruption is defined as the premature separation of normally situated placenta before delivery. It occurs in about 0.6 -1 % of all births.

Etiology are hypertensive disorders of pregnancy, preterm premature rupture of membranes, multiple pregnancy, hydramnios, thrombophilias, fibroids complicating pregnancy , trauma.

Abruption has been associated with low socio economic status, multiparity , smoking, cocaine use . Folic acid deficiency has been linked to abruption.

Various types of abruption are concealed, revealed and mixed ones.

Patho physiology

Lack of trophoblastic invasion leading to decreased placental blood flow results in spasm of blood vessels ,anoxia and endothelial disruption causing bleeding. Other causes genetic predisposition, vascular malformation and placental abnormalities.

RCAS1 , membrane protein involved in maternal immune tolerance plays a major role in the mechanism of abruption. It may present as a retroplacental haematoma to couvelaire uterus.Extravasated blood may result in atonic uterus and also the thromboplastins from the haematoma will enter into maternal circulation resulting in disseminated intravascular coagulation.

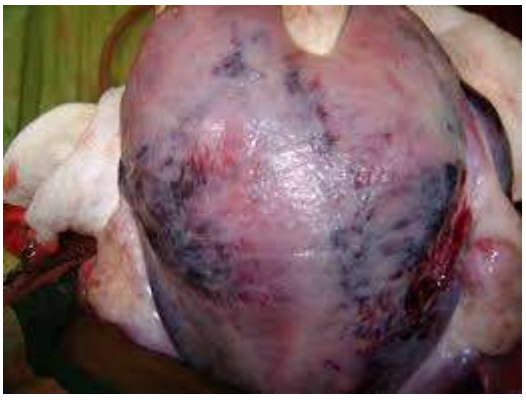


FIG NO 6 COUVELAIRE UTERUS

Sudden ischaemia will result in bilateral cortical necrosis and acute tubular necrosis of the kidneys.

Symptoms include insignificant bleeding , abdominal pain, fetal demise and coexisting eclampsia features.

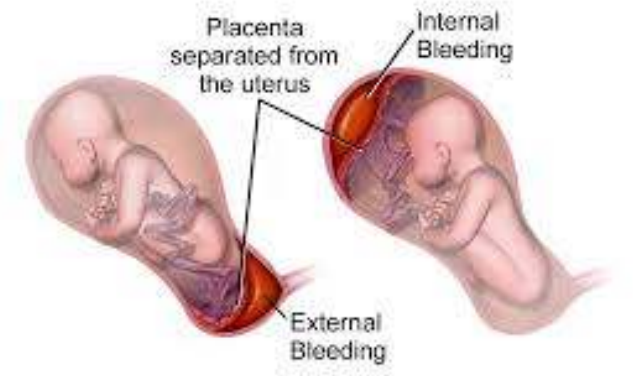


FIG NO 7 PLACENTAL ABRUPTION

Uterine size will be more than the gestational age and there may be retroplacental clot between the placental margin .In some cases blood may seep in to myometrium leading to the formation of couvalaire uterus.

FIG NO8 COUVELAIRE UTERUS (2)

Grading of abruption

Grade 0 Diagnosed incidentally after delivery.

Grade 1 Slight vaginal bleeding and uterine irritability,no maternal and fetal compromise.

Grade 2 Mild to moderate bleeding, maternal tachycardia, hypotension , fetal distress, decreased fibrinogen levels.

Grade 3 Severe haemorrhage with tetanic and painful uterus leading to shock and fetal demise with signs of coagulopathy.

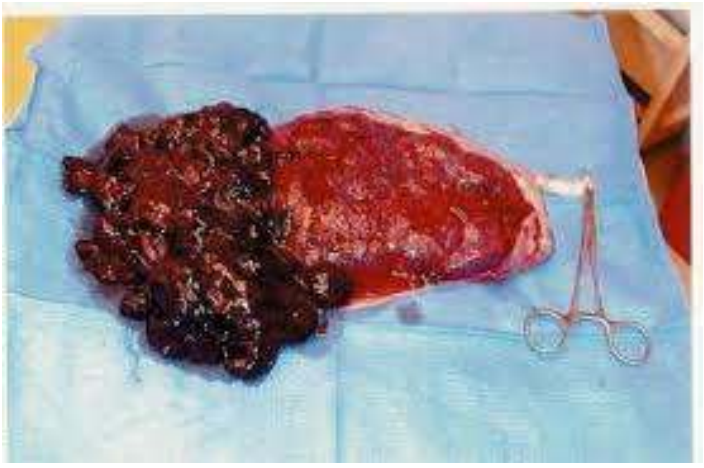


FIG NO 9 RETROPLACENTAL CLOT IN ABRUPTION

Complications include obstetric haemorrhage ,need for blood transfusions, hysterectomy for atonicity, disseminated intravascular coagulation, renal failure, sepsis, pulmonary edema, postpartum anaemia, and also maternal death.

Perinatal complications include low birth weight, preterm delivery, asphyxia and perinatal deaths.

Management

Blood and fluids is replaced to prevent hypovolemia and anaemia.

Urine output is to be maintained atleast 30 ml/hr. Other supportive measures should be carried out.

Management depends on the individual case , gestational age and severity. Whenever fetal demise occurs, vaginal delivery is preferable. In case of maternal and fetal compromise, caesarean delivery is indicated.

HYPERTENSIVE DISORDERS OF PREGNANCY

Pre eclampsia comprises a triad of hypertension beyond 20 weeks, edema and proteinuria > 300 mg protein in a 24 hour urinary protein. About 5-7% of pregnant patients fall into this criteria.[1-4]

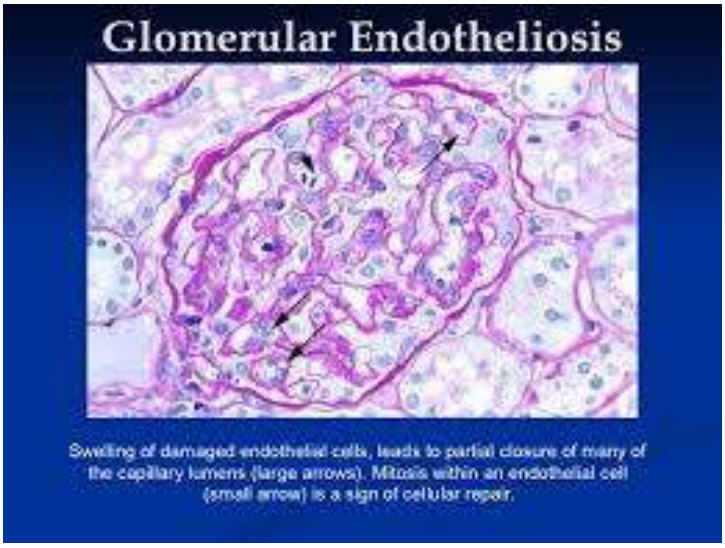
Eclampsia is a condition where seizures occurs in a pre eclamptic patient.[5,6]

RISK FACTORS

The various risk factors for preeclampsia are nulliparity , multiple gestation, molar pregnancy , history of renal disease , chronic hypertension, pregestational diabetes mellitus, obesity , rheumatic disease, antiphospholipid antibody and age over 40 years.

PATHOLOGY

In preeclampsia, renal perfusion and glomerular filtration will be reduced as a result of increased renal afferent arteriolar resistance. There will be glomerular endotheliosis. Decreased filtration leads to increased creatinine levels (> 1 mg/dl).



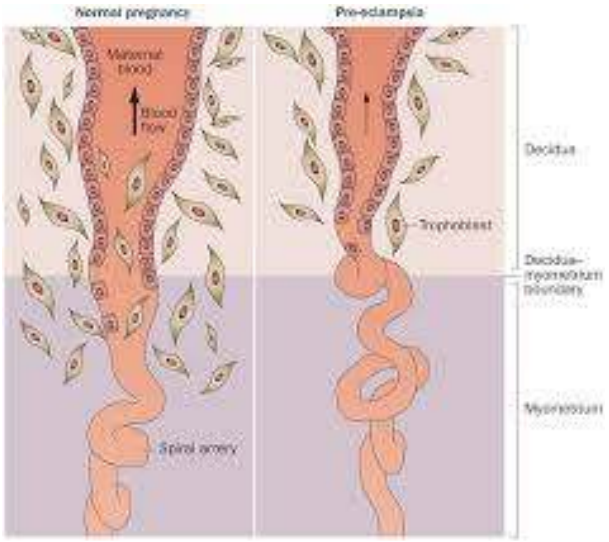


FIG NO10 &11 PATHOLOGY OF PREECLAMPSIA

Levels wil normalize after 10 days in most of the cases.

Uric acid is also elevated in most of the cases. This is because of reduced glomerular filtration and increased tubular reabsorption.

There will be diminished calcium excretion due to increased tubular reabsorption.

Acute tubular necrosis occurs in these cases.

CLINICAL PRESENTATION

Signs of preeclampsia are raised blood pressure >140/90 mm Hg , proteinuria, generalized edema including the face and hands, headache, visual disturbances upper abdominal pain , oliguria, fetal growth restriction, pulmonary odema, reduced fetal movements and decreased platelet count.

In severe preeclampsia and eclampsia , there is widespread spasm of the arterioles leading to multi organ failure which endanger maternal and fetal lives leading to death sometimes.

Maternal complications of preeclampsia are eclampsia, cerebral hemorrhage, thrombosis, cerebral edema ,acute renal failure , aspiration bronchopneumonia, pulmonary odema, HELLP syndrome, DIVC leading to hemorrhage and death.[7]

Neonatal complications may include IUGR, still birth and neonatal asphyxia.

MANAGEMENT

Management depends on the various factors like gestational age, severity of hypertension and end organ damage. If the gestational age is less than 34 weeks, expectant management is indicated along with corticosteroids for lung maturity. Maternal stabilization is done in the following cases like uncontrolled hypertension, eclampsia, pulmonary edema, placental abruption, DIVC, non reassuring fetal heart tracing and fetal distress. Delivery of the fetus is the immediate management along with antihypertensives, magnesium sulphate and maintaining fluid balance.

HELLP SYNDROME

Severe preeclampsia in about 2 to 12 % of cases will be accompanied by Hemolysis (H) , Elevated Liver enzymes (EL) , and low platelet count (LP) , HELLP SYNDROME.

Diagnostic criteria for HELLP Syndrome

Hemolysis Abnormal peripheral smear

Elevated liver enzymes AST>70 / L LDH>600 U/L Increased indirect bilirubin

Low platelets <1,50,000

HELLP syndrome may be subdivided in to three types based on the platelet count.

Severe – class 1 platelets <50,000

Moderate – class 2 platelets 50,000 to 99,000

Mild – class 3 platelets >1 lakh

ETIOLOGY

Pathology is due to microangiopathic hemolytic anaemia along with vascular endothelial injury leading to platelet consumption , resulting in small to diffuse haemorrhage into liver capsule causing hematoma, capsular tears and intra peritoneal bleeding. Factor V Leiden and placental CD95 is associated with HELLP Syndrome.[40]

CLINICAL FEATURES AND DIAGNOSIS

Upper abdominal pain, tenderness, nausea, vomiting, malaise, headache , edema , weight gain , hypertension and proteinuria.

Diagnosis is by above criteria. There is also some overlap between HELLP syndrome and AFLP. HUS and TTP also have similar presentations.

MANAGEMENT

Majority of patients recover soon after delivery within six days. Life threatening complications need specific treatment with plasmapheresis, plasma volume expanders, anti thrombotic agents, steroids , fresh frozen plasma and dialysis. Delayed recovery is observed in severe preeclamptic patients with renal failure in antenatal period. These patients are managed with dialysis in acute phase itself. The recent publications have shown a maternal mortality of around 1% with raised perinatal mortality due to early gestational age.

SEPSIS

Acute kidney injury in septic abortion is common in India due to illegal abortion. Also occurs in UTI leading to pyelonephritis. Mortality rate is about 30-65%. If there is no improvement in 24 hours, emergency hysterectomy is done .

Study by Barlelett and Yahia has shown a survival rate of about 100% after hysterectomy.

Mechanism of AKI occurs in two ways one is by endotoxemia causing swelling and damage of glomerular endothelial cells resulting in hypovolemia. Second one is by platelet aggregation and lysis to activation of coagulation factors leading to DIVC.

Features will be hemolytic anaemia, thrombocytopenia, decreased GFR, raised creatinine, ARDS and shock.

Management is by higher antibiotics, fluid replacement and if needed positive pressure ventilation in severe cases.

ACUTE FATTY LIVER OF PREGNANCY

It is a sudden deranging illness occurring most commonly in the third trimester due to micro vesicular fatty infiltration resulting in encephalopathy and hepatic failure.[31] Incidence is about 1 in 14,000 pregnancies.[32,33]

Etiology of acute fatty liver of pregnancy is due to abnormalities in intra mitochondrial fatty acid oxidation.[34] Beta oxidation of fatty acid in hepatic mitochondria requires several enzymes.

Mitochondrial trifunctional protein and its alpha sub unit long chain 3-hydroxyacyl –CoA dehydrogenase (LCHAD) are 2 main enzymes and the genetic mutation in LCHAD is closely associated with AFLP.

The presentation varies from asymptomatic to fulminant hepatic failure. Typical features include a period of 1 to 2 weeks anorexia, headache, nausea, malaise, right upper quadrant pain, jaundice, hypertension, ascites, renal failure and hepatic encephalopathy. Most of the patients will have associated preeclampsia and HELLP syndrome.[37]

These patients present with elevated bilirubin levels , anaemia, elevated liver enzymes , high WBCcount, normal or low platelets, hypoglycemia, renal dysfunction, coagulopathy with or without DIVC.

Plasma urea, uric acid and creatinine levels are usually elevated. Uric acid levels may increase days before the symptoms appear.[38] This is the clinching point for diagnosing acute fatty liver of pregnancy.[39]

Diagnosis is made only presumptively by clinical and biochemical diagnosis. Confirmatory test is by histological diagnosis of microvesicular fatty infiltration in zone 3 and portal inflammation with cholestasis .3

Differential diagnosis are fulminant hepatic failure and HELLP syndrome.

Management is only immediate termination of pregnancy and intensive supportive care is essential for maternal and fetal survival.

INR of 1.5 and platelet count of > 50,000 should be maintained and prophylactic antibiotics to prevent infection are given.

Infections and bleeding complications are the most life threatening complications.

AMNIOTIC FLUID EMBOLISM

This is one of the rare complication occurring in pregnant patients where amniotic fluid, debris, fetal cells enter the maternal circulation causing sudden collapse of the patients. Risk factors associated with this

condition are older age, induction of labour, placental abnormalities, eclampsia , polyhydramnios , cervical lacerations and uterine rupture.

Pulmonary artery vasospasm occurs. There will be raised right ventricular pressure. Hypoxia occurs due to myocardial and pulmonary capillary damage leading to left heart failure, acute respiratory distress , massive haemorrhage , uterine atony and DIVC.

Immediate administration of oxygen, intravenous fluids ,replacement of blood and blood products, uterine artery embolisation, hysterectomy in cases of severe haemorrhage. AKI will be seen in patients surviving the acute attack.

DISSEMINATED INTRA VASCULAR COAGULATION

Thrombo haemorrhagic disorder occurring in patients with the following conditions like abruption, intrauterine fetal death , amniotic fluid embolism, septic abortion, molar pregnancy, severe haemorrhage , severe preeclampsia , AFLP , HELLP syndrome and puerperal sepsis. It is also called as consumptive coagulopathy due to activation of aberrant coagulation cascade.

Imbalance between clotting and unclotting mechanism is seen. Thromboplastins are released resulting in activation of both extrinsic and intrinsic pathways. Coagulation factors are depleted leading to uncontrolled haemorrhage.

The most common cause of DIVC is abruption placenta accounting for about 10%. Clinical features will be signs of primary condition , petechiae, purpura, haematoma , ecchymosis ,bleeding from wound site , venepuncture sites , AKI, shock and acidosis.

Treatment includes correction of the underlying cause , other supportive measures like replacement of blood products to maintain circulatory blood volume and to prevent end organ damage.

THROMBOTIC MICROANGIOPATHIES

This constitutes thrombotic thrombocytopenic purpura and hemolytic ureamic syndrome. TTP will present with thrombocytopenia, fever, neurological abnormalities, renal impairment and hemolytic anaemia . HUS will present with more renal involvement and few neurological abnormalities. It is due to the deficiency of ADAMTS -13 gene.HUS is mostly due to endothelial damage to viral and bacterial infections .

These patients will present with thrombocytopenia, fragmentation hemolysis and end organ damage. Headache , altered level of consciousness, seizures and stroke wil occur. Renal failure is more prominent in HUS. Most of the patients will require dialysis.

Biochemical parameters altered are as follows. Low platelet counts, hemolytic blood picture of peripheral smear with schizocytes fragmented RBCs, increased reticulocytes, increased lactate dehydrogenases, and decreased haptoglobin will be present.

Treatment will be plasmapheresis with fresh frozen plasma transfusions. Severe cases will have renal failure and often need dialysis and even transplantation of kidneys . Maternal mortality is very high in these conditions.

MANAGEMENT

Management of acute kidney injury in pregnancy depends on the understanding of the physiological changes occurring during pregnancy and early identification and prompt management. Basic principle in treating renal failure comprise the following like restoration of intra vascular volume , treating the underlying condition and deranged electrolyte abnormalities should be corrected.Treatment lies at correcting the primary cause for acute kidney injury. In most of the cases , delivery

of the fetus remains the first management. Severe cases may require dialysis (hemodialysis) . Co existing anaemia should be corrected . Blood loss should be corrected and replaced . Infections should be treated with adequate antibiotics . If surgical cause remains, it should be corrected by surgical interventions.

AZAR MEHRABADI et al studied the obstetric renal failure incidence in postpartum haemorrhage, hypertensive disorders of pregnancy and the other risk factors in Canada. The study showed an increase in patients with acute renal failure presenting with hypertensive disorders from 1.66 to 2.68 per 10,000 deliveries.[12,17]

KALIKI HYMAVATHI REDDY et al reported a case study of preeclampsia leading to acute renal failure intervened at the right time leading to recovery from the illness.[8,9]

In the study by VIJAY BHARGAVA et al incidence of acute renal failure ranged about 5.66%, most common pathology being pre eclampsia and eclampsia followed by sepsis.[28-30]

In the study by PRAKASH et al incidence has declined in the past few years due to the availability of antenatal care, safe abortion practice, decreased illegal abortion and wide spread use of higher antibiotics to combat infections.[1-7]

SMITH et al 1965, KENNEDY et al 1973 , ALI et al 1973 have reported that the major cause of acute kidney injury in obstetrics were due to antepartum and postpartum haemorrhage.[20]

CENGIZ UTAS et al in his study evaluated the etiological spectrum of acute kidney injury in 2000 in the developing countries. They concluded that surgical causes leading to acute kidney injury has declined. Medical cause began to predominate nowadays.[10,30]

MOHAMMED IRFAN KHATTAK et al studied the various etiological factors contributing to renal failure. The most common cause according to him is sepsis.[42]

NAMRATA KHANAL et al in his study found that increase in the maternal mortality is associated with increasing age, increased severity of the presentation of the illness, any associated chronic renal illness and the need for ionotropes and assisted ventilator support. Good antenatal care, early identification and correction of problem leading to acute kidney injury by appropriate tertiary care management will reduce the incidence of acute kidney injury in pregnancy.[22,26,28]

KILARI SUNIL KUMAR et al studied the clinical profile, management and the outcome of renal failure in pregnancy. In his study, patients of about 4.24% contribute to acute kidney injury and most prevalent in the postpartum perioddue to sepsis.[25-32]

SHAMIMUR RAHMAN et al from Bangladesh studied the incidence of renal failure in the developing countries and found to be more in the multigravidas in the third trimester and with irregular antenatal care patients.[41]

A observational prospective study by MOHAMMED ARRAYHANI et al showed the characteristics and the factors associated with the unfavourable outcome, preeclampsia and eclampsia being the most commonest cause.[10-12] HELLP syndrome is associated with higher incidence of maternal morbidity and mortality. [21-24]

MATERIALS AND METHODS

Thirty four women with acute kidney injury admitted and treated at Coimbatore medical college hospital , Coimbatore from July 2014 to July 2015 were studied .

A detailed history including th patient's age ,socio economic status, booking, parity and details of menstrual history to arrive at the expected date of delivery was obtained.

Patients were enquired in detail about their complaints like vomiting , bleeding per vaginum either during the antenatal or in the postnatal period , headache, decreased urine output, edema of both legs , facial puffiness, fever, yellow coloured urine, seizures and others.

Past history of anaemia , intra uterine fetal death , preeclampsia ,abortion, fever, molar pregnancy ,treatment for infertility, blood transfusion etc., are obtained. Co morbid conditions like anaemia , chronic hypertension, renal disease, pregestational hypertension , hypothyroid , obesity and heart disease are ruled out. Systemic and obstetric examinations are carried out.

Investigations included complete blood count, renal function tests, liver function tests , serum uric acid, urine analysis , 24 hours urinary protein , peripheral smear report , ultrasound obstetrics with maternal organs were carried out as and when required.

HIV screening was done for all patients.

Nephrologist opinion was obtained for all the patients.

Fundus examination was done for suitable cases.

For antenatal cases, labour was closely monitored and if indicated LSCS was done for some of the cases. If in severe renal failure, early termination of pregnancy and hemodialysis was preferred. Blood transfusion was given for indicated cases. Some patients require ventilator support also.

Patients were kept in the labour ward for close observation. Renal function tests were repeated as and when required and hemodialysis was planned accordingly.

Soon after delivery , babies were assessed by the Paediatrician. Alive or dead , term or preterm ,sex, gestational age at birth , weight of the baby, APGAR score and presence of any congenital anomalies were

looked for. If preterm babies were admitted in NICU and were given further care.

The maternal outcome was noted in terms of mode of delivery , maternal complications and the mortality . the relation of maternal morbidity and mortality to the admission serum creatinine levels were analysed.

To identify the various etiologies and the distribution of acute kidney injury in relation to age , parity and trimesters.

Fetal outcome was studied by perinatal morbidity and mortality.

Study design: Prospective cohort study

Study population: 34 patients

Inclusion criteria: All acute kidney injury pregnant patients admitted to labour ward in Obstetrics and Gynaecology department in Coimbatore medical college , Coimbatore.

Exclusion criteria:

Previous history of hypertension, diabetes , history of renal disease, ultrasound finding of renal scarring, small kidneys , increased serum creatinine levels before pregnancy.

OBSERVATION AND RESULTS

The incidence of acute kidney injury is about 1 in 15,000 to 20,000 deliveries . A prospective study of all antenatal patients admitted to Coimbatore Medical College Hospital, Coimbatore with acute kidney injury was evaluated from the month of July 2014 to July 2015. Total number of admissions during this period were 11,389. According to this study , the incidence of acute kidney injury is 4.6 per 1000 deliveries.

Prakash et al reported an incidence of about 5% in India. Mehrabadi Aet al reported that there is an increased incidence of obstetric kidney injury attributed to hypertensive disorders of pregnancy to about 2.68 per 10,000 deliveries.

AGE GROUP:

Out of 34 women studied , 47 % were in the age group of 21 to 25 years. (TABLE 1) .

PARITY AND GESTATIONAL AGE:

About 21% of the women of 34 were third gravid. The incidence is more common in the third trimester and accounts to about 44%. 65% of the women fall in antenatal period. (TABLE 2, 3, 4).

ETIOLOGY OF ACUTE KIDNEY INJURY:

Out of 34 patients , 20 had hypertensive disorders of pregnancy (58%), 4 had post partum haemorrhage(12%) , 1 had abruptio placenta (3%), 1 had HELLP syndrome(3%) , 3 had anaemia(9%).(TABLE 6).

MODE OF DELIVERY:

Out of 34 patients, 16 delivered by Lower segment caesarean section (47%) ,14 by labour natural (41%). (TABLE 7).

COMPLICATIONS:

Out of 34 women, 21 patients recovered without any major complications. Out of 21 patients , 10 required dialysis(48%).

13 patients died of complications due to pulmonary edema(3), hemolytic uremic syndrome with cardiac arrest(2) ,DIVC (4) ,ARDS(2) and CVT(2).

MATERNAL OUTCOME:

Among 34 patients , 22 were antenatal and 12 were postnatal . Out of the 13 deaths, dialysis was done for 7 patients. 6 patients died due to hemodynamic instability.(TABLE 19%)

FETAL OUTCOME

Out of 34 , 21 were live births(62%) , 11 were dead(32%) and abortion (2%) . Out of 21 , 14 were term (67%) .

RESULT AND ANALYSIS

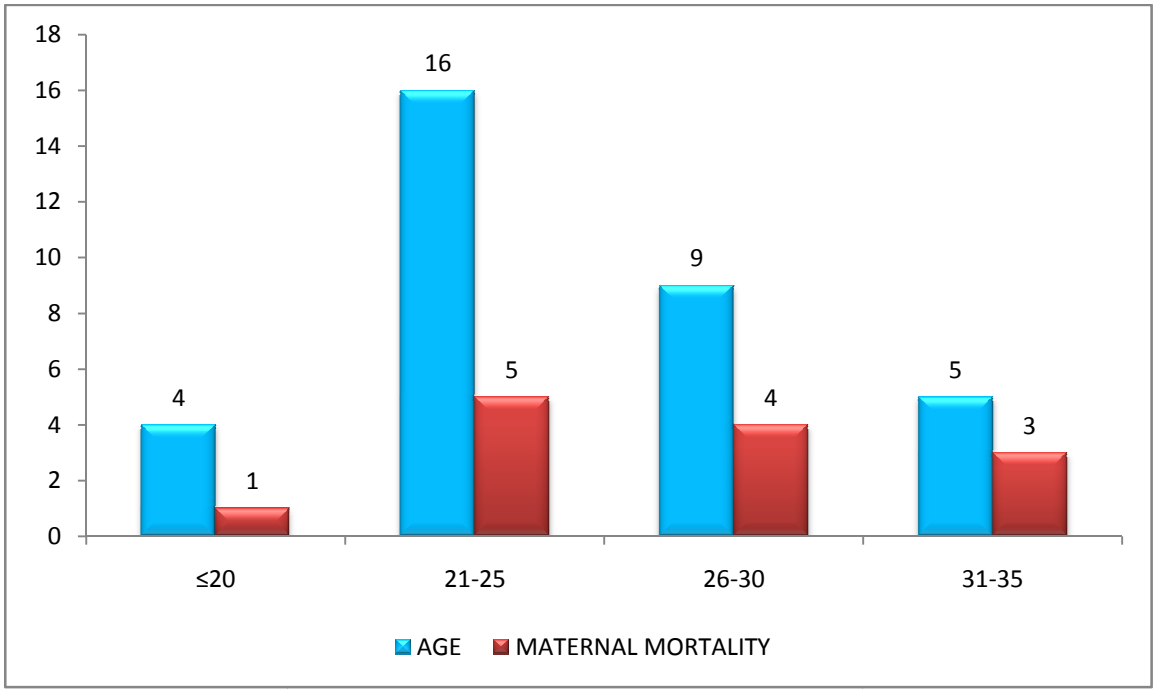
TABLE 1

AGE DISTRIBUTION

AGE	FREQUENCY	MATERNAL MORTALITY
≤20	4	1
21-25	16	5
26-30	9	4
31-35	5	3

CHART 1

AGE DISTRIBUTION



Out of 34 patients studied 47% were in the age group of 21-35

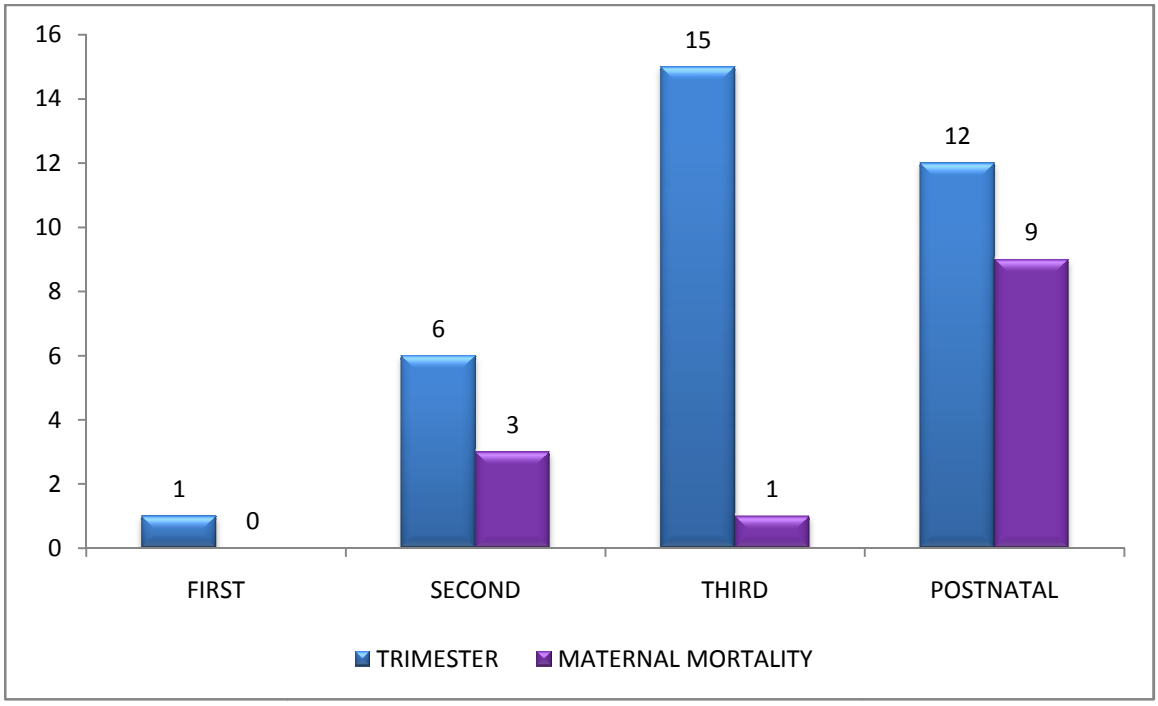
TABLE 2

TRIMESTER DISTRIBUTION

TRIMESTER	FREQUENCY	MATERNAL MORTALITY
FIRST	1	0
SECOND	6	3
THIRD	15	1
POSTNATAL	12	9

CHART 2

TRIMESTER DISTRIBUTION



Out of the 34 patients studied 44% were in the third trimester. High mortality is seen in the postnatal period.

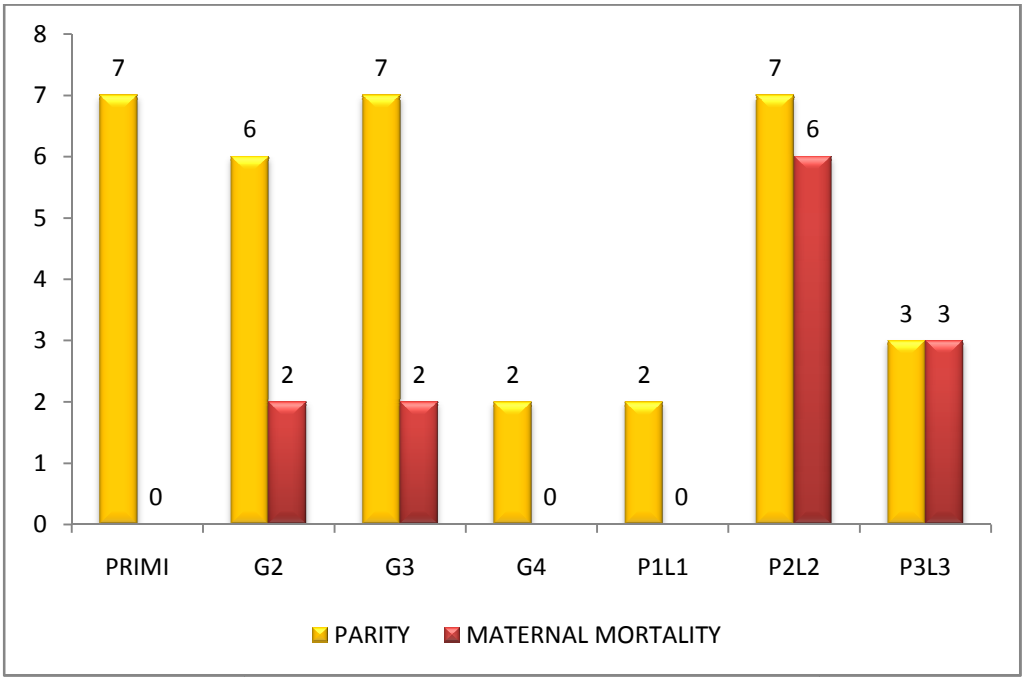
TABLE 3

PARITY DISTRIBUTION

PARITY	FREQUENCY	MATERNAL MORTALITY
ANTENATAL		
PRIMI	7	0
G2	6	2
G3	7	2
G4	2	0
POSTNATAL		
P1L1	2	0
P2L2	7	6
P3L3	3	3

CHART 3

PARITY DISTRIBUTION



Out of the 34 patients studied, multigravida constituted to about 35%

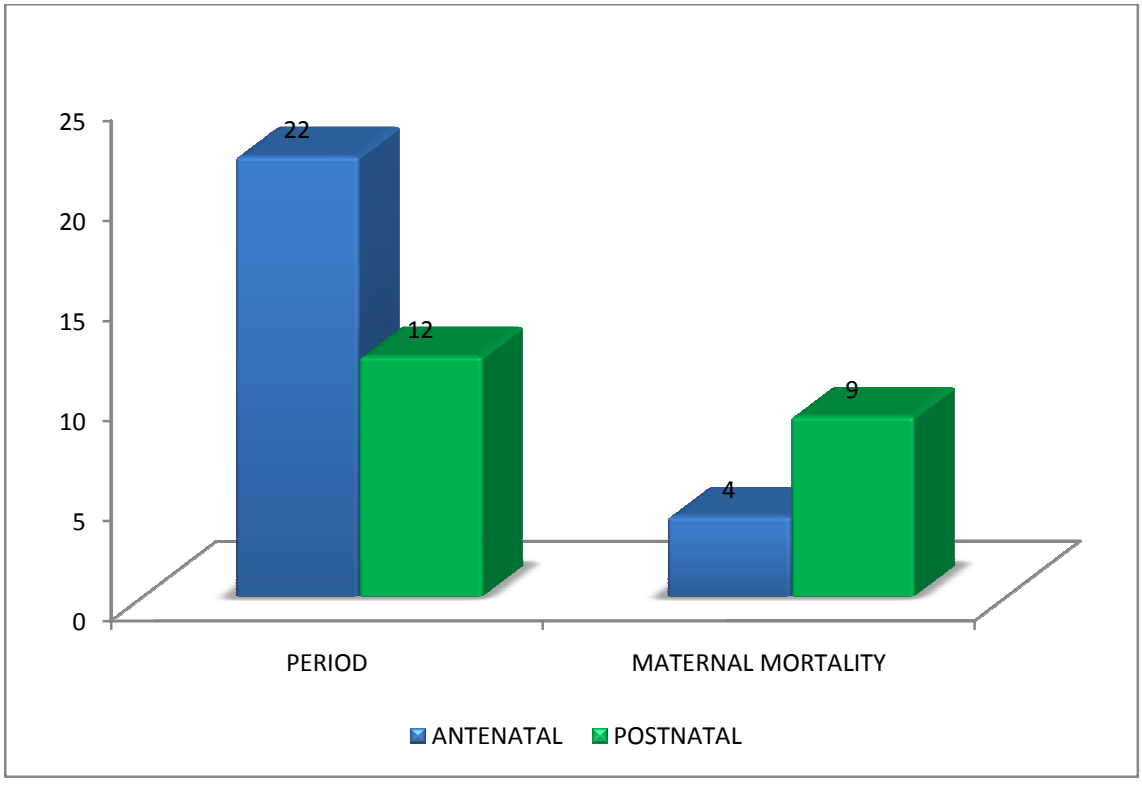
TABLE 4

PERIOD OF PRESENTATION OF ACUTE KIDNEY INJURY

PERIOD	FREQUENCY	MATERNAL MORTALITY
ANTENATAL	22	4
POSTNATAL	12	9

CHART 4

PERIOD OF PRESENTATION OF ACUTE KIDNEY INJURY



Out of 34 patients studied, 65% were in their antenatal period

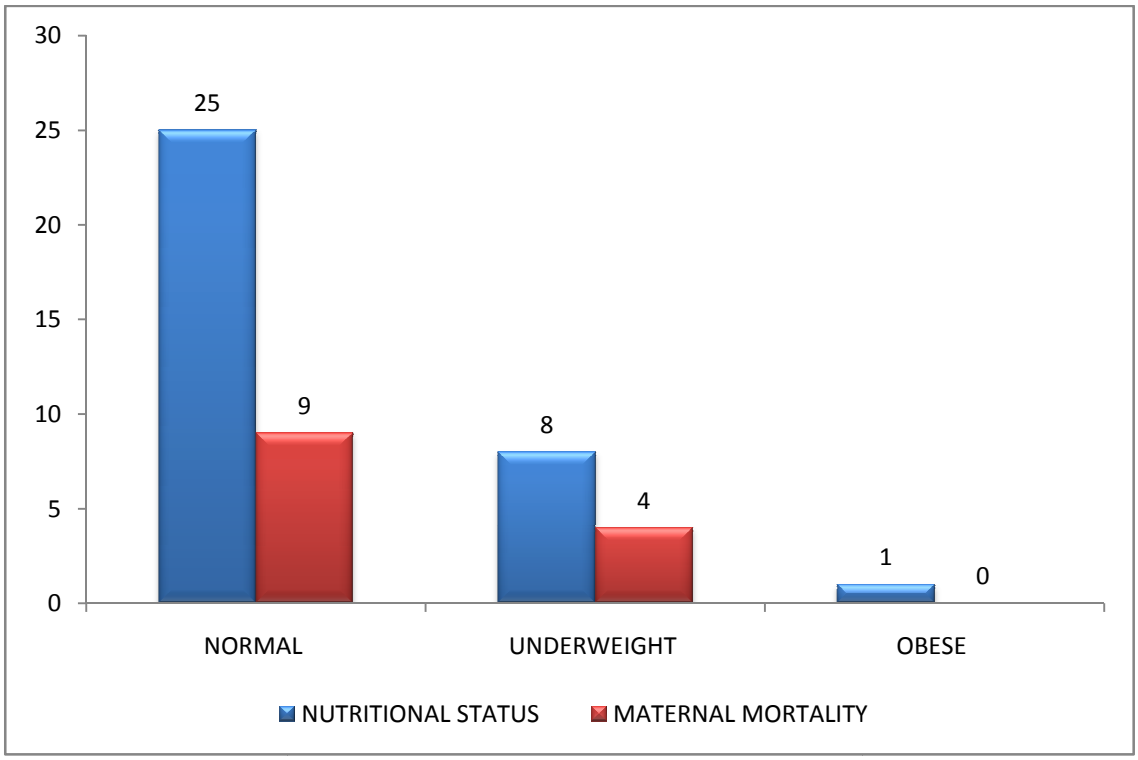
TABLE 5

NUTRITIONAL STATUS

NUTRITIONAL STATUS	FREQUENCY	MATERNAL MORTALITY
NORMAL	25	9
UNDERWEIGHT	8	4
OBESE	1	0

CHART 5

NUTRITIONAL STATUS



Out of 34 patients, 74% were in the normal limits of BMI.

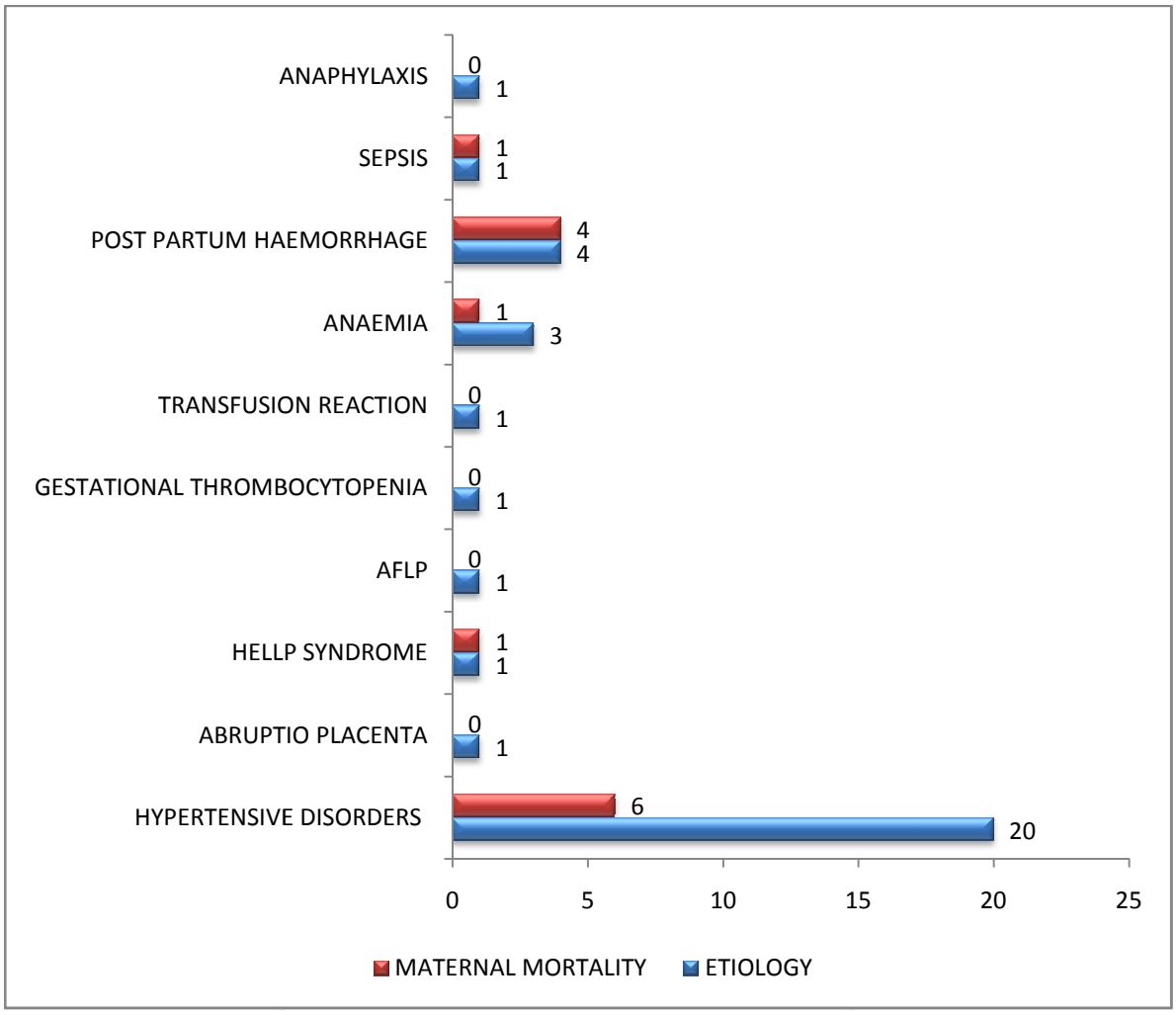
TABLE 6

ETIOLOGY OF ACUTE KIDNEY INJURY

ETIOLOGY	FREQUENCY	MATERNAL MORTALITY
HYPERTENSIVE DISORDERS	20	6
ABRUPTIO PLACENTA	1	0
HELLP SYNDROME	1	1
AFLP	1	0
GESTATIONAL THROMBOCYTOPENIA	1	0
TRANSFUSION REACTION	1	0
ANAEMIA	3	1
POST PARTUM HAEMORRHAGE	4	4
SEPSIS	1	1
ANAPHYLAXIS	1	0

CHART 6

ETIOLOGY OF ACUTE KIDNEY INJURY



Out of 34 patients studied, 58% had hypertensive disorders of pregnancy. Out of 34 patients , 3 patients had antepartum eclampsia and 2 developed post partum seizures.

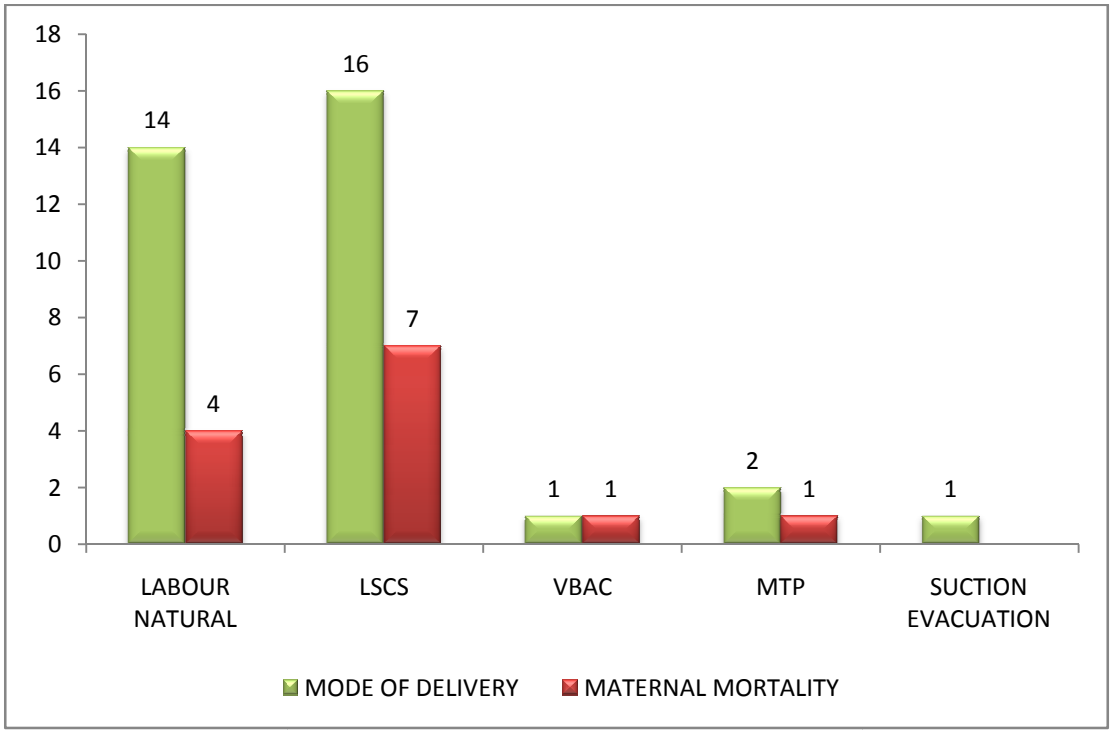
TABLE 7

MODE OF DELIVERY

MODE OF DELIVERY	FREQUENCY	MATERNAL MORTALITY
LABOUR NATURAL	14	4
LSCS	16	7
VBAC	1	1
MTP	2	1
SUCTION EVACUATION	1	

CHART 7

MODE OF DELIVERY



Out of 34 patients, 47% delivered via labour natural

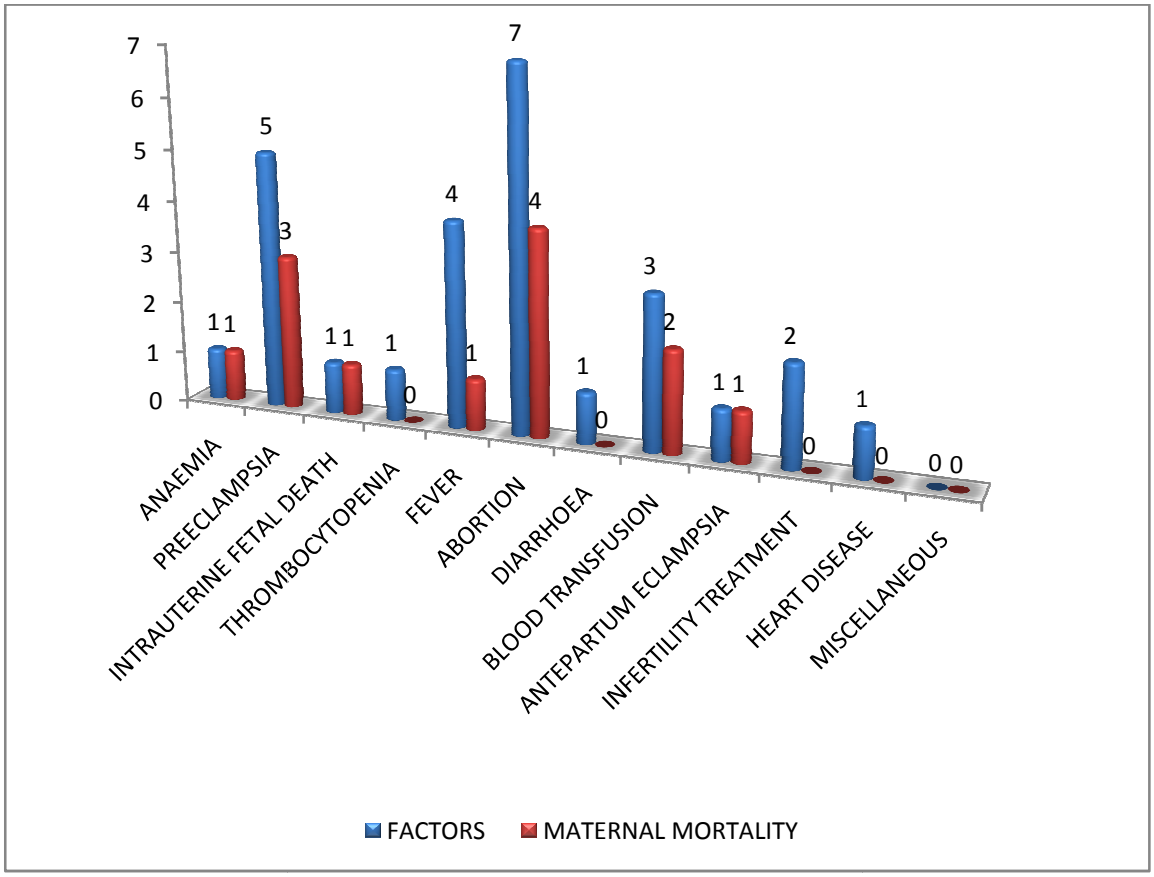
TABLE 8

ASSOCIATION OF OTHER FACTORS IN THE PAST

FACTORS	FREQUENCY	MATERNAL MORTALITY
ANAEMIA	1	1
PREECLAMPSIA	5	3
INTRAUTERINE FETAL DEATH	1	1
THROMBOCYTOPENIA	1	0
FEVER	4	1
ABORTION	7	4
DIARRHOEA	1	0
BLOOD TRANSFUSION	3	2
ANTEPARTUM ECLAMPSIA	1	1
INFERTILITY TREATMENT	2	0
HEART DISEASE	1	0
MISCELLANEOUS	0	0

CHART 8

ASSOCIATION OF OTHER FACTORS IN THE PAST



Out of 34 patients studied, 21% had previous history of abortion

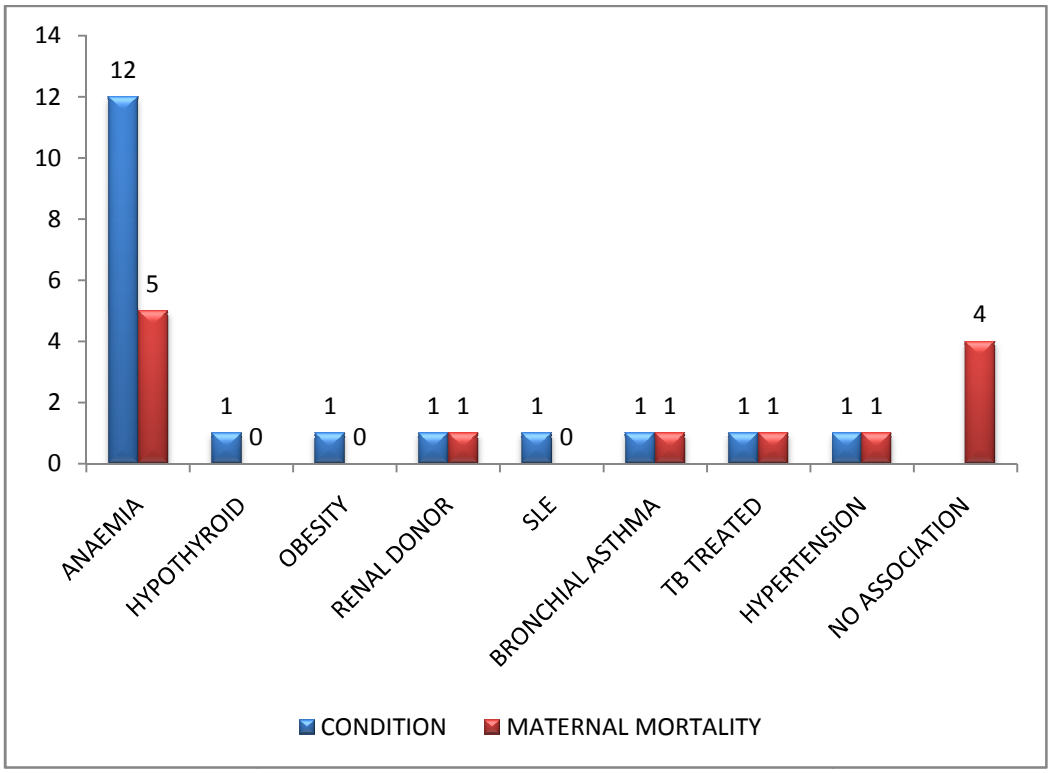
TABLE 9

ASSOCIATION WITH CO MORBID CONDITION

CONDITION	FREQUENCY	MATERNAL MORTALITY
ANAEMIA	12	5
HYPOTHYROID	1	0
OBESITY	1	0
RENAL DONOR	1	1
SLE	1	0
BRONCHIAL ASTHMA	1	1
TB TREATED	1	1
HYPERTENSION	1	1
NO ASSOCIATION		4

CHART 9

ASSOCIATION WITH CO MORBID CONDITION



Out of 34 patients, anaemia is the most common associated comorbid condition (12 cases)

TABLE 10

EDEMA

EDEMA	FREQUENCY
PRESENT	24
ABSENT	10

CHART 10

EDEMA

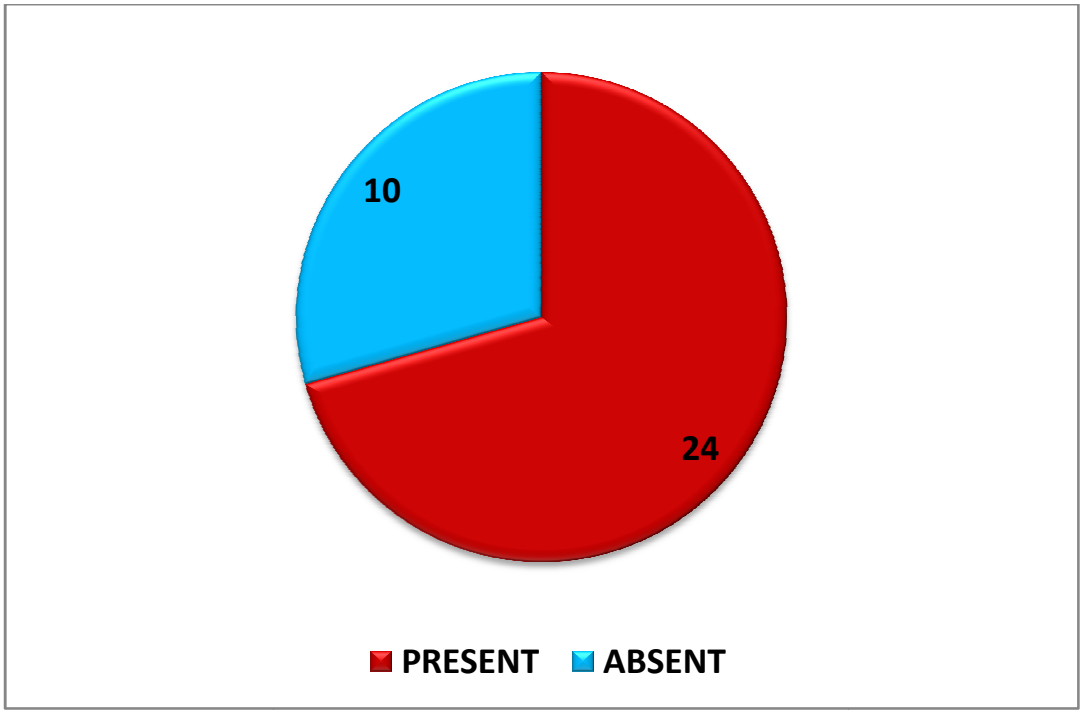


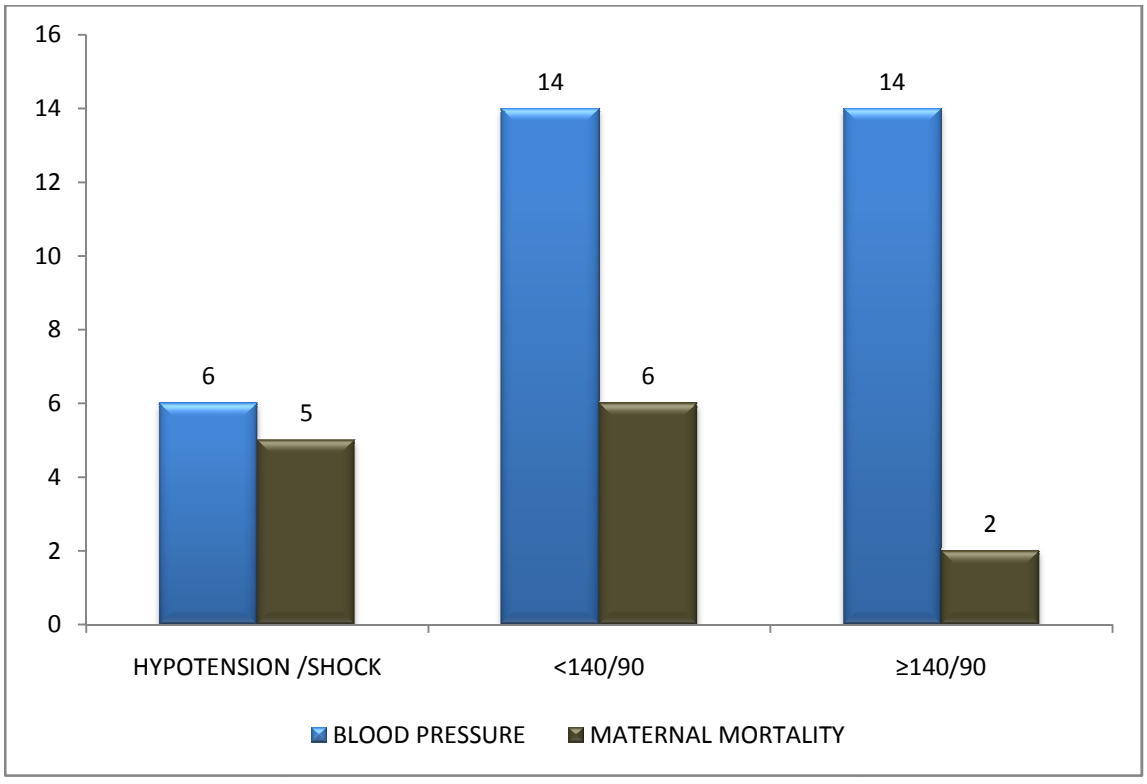
TABLE 11

INITIAL BLOOD PRESSURE AT PRESENTATION

BLOOD PRESSURE	FREQUENCY	MATERNAL MORTALITY
HYPOTENSION /SHOCK	6	5
<140/90	14	6
≥140/90	14	2

CHART 11

INITIAL BLOOD PRESSURE AT PRESENTATION



Shock carries a very bad prognosis.

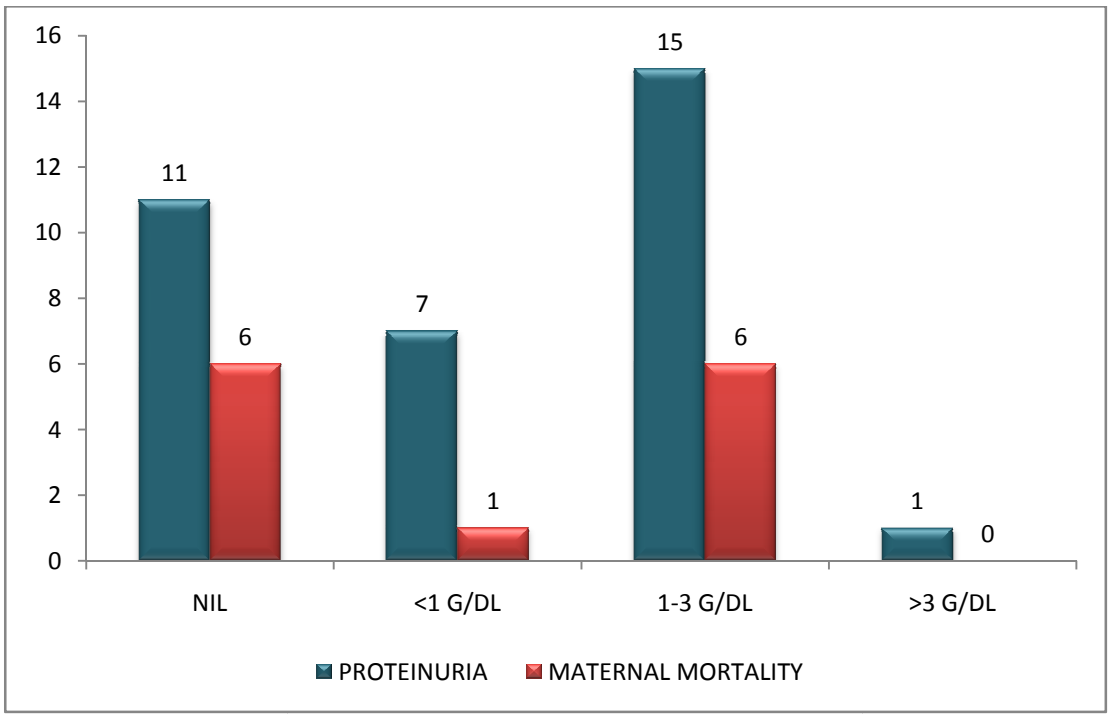
TABLE 12

PROTEINURIA

PROTEINURIA	FREQUENCY	MATERNAL MORTALITY
NIL	11	6
<1 G/DL	7	1
1-3 G/DL	15	6
>3 G/DL	1	0

CHART 12

PROTEINURIA



Proteinuria does not correlate with the prognosis.

TABLE 13

LEVEL OF INITIAL UREA WITH ACUTE KIDNEY INJURY

UREA	FREQUENCY	MATERNAL MORTALITY
<40	8	1
40-100	20	9
100-200	3	3
>200	3	0

CHART 13

LEVEL OF INITIAL UREA WITH ACUTE KIDNEY INJURY

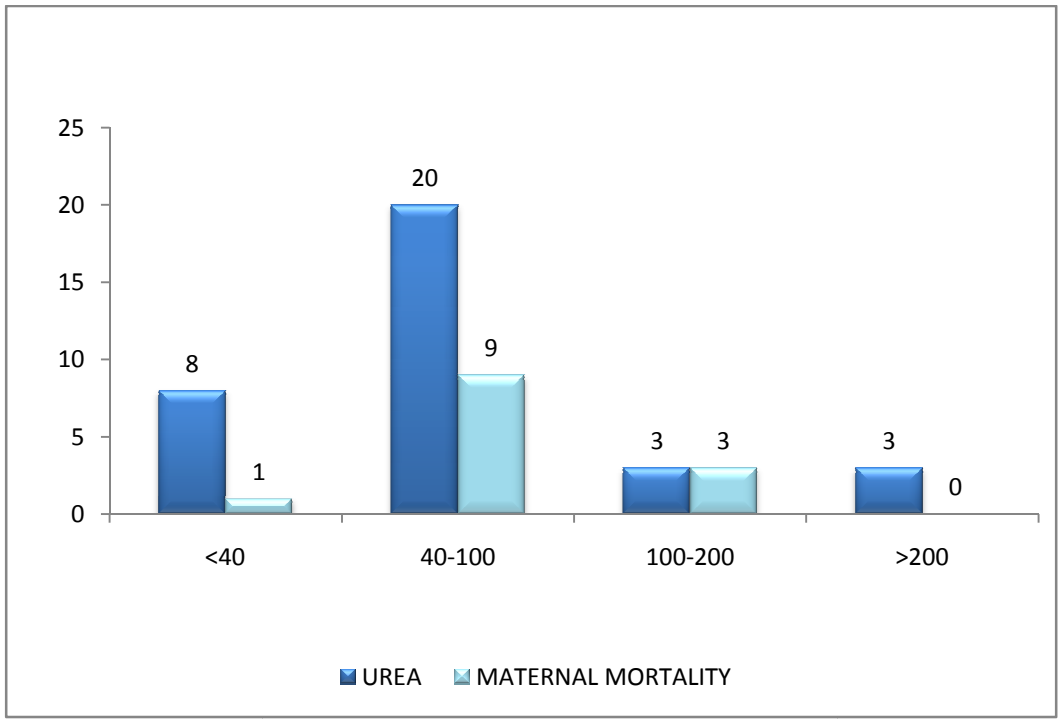


TABLE 14

DIURESIS

DIURESIS	**FREQUENCY**	**MATERNAL MORTALITY**
NONOLIGURIC	11	4
OLIGURIA	19	6
ANURIA	4	3

CHART 14

DIURESIS

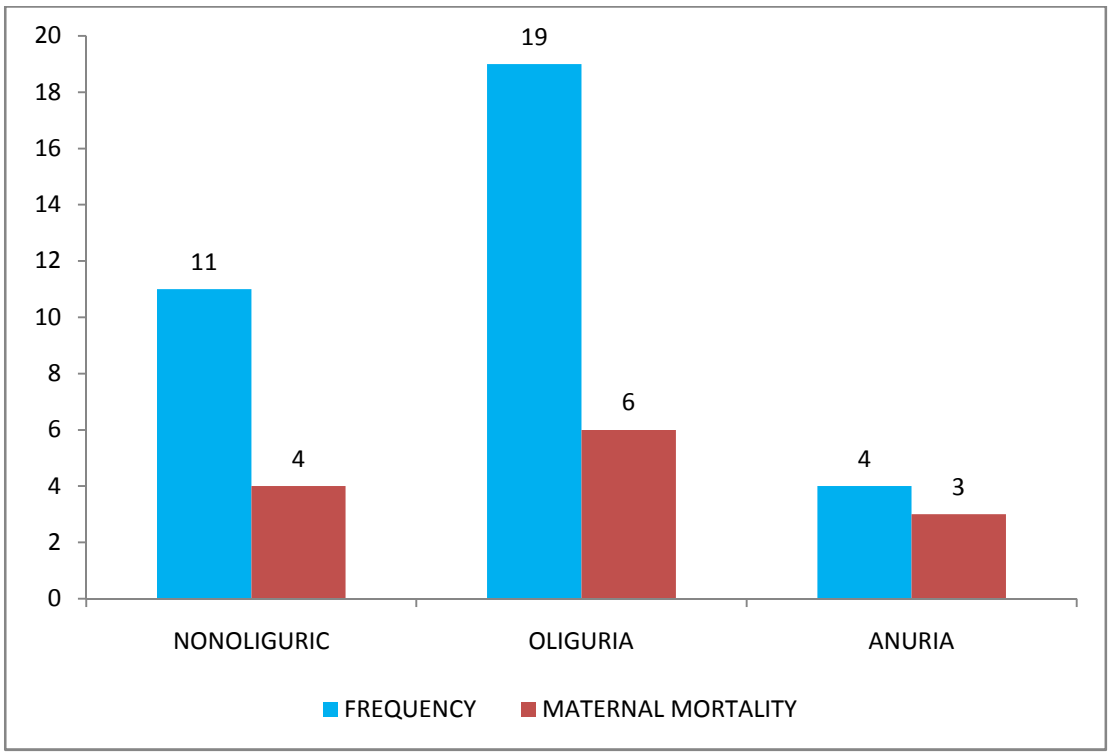


TABLE 15

LEVEL OF INITIAL CREATININE WITH ACUTE KIDNEY INJURY

CREATININE	FREQUENCY	MATERNAL MORTALITY
<1	1	0
1-5	11	10
>5	22	3

CHART 15

LEVEL OF INITIAL CREATININE WITH ACUTE KIDNEY INJURY

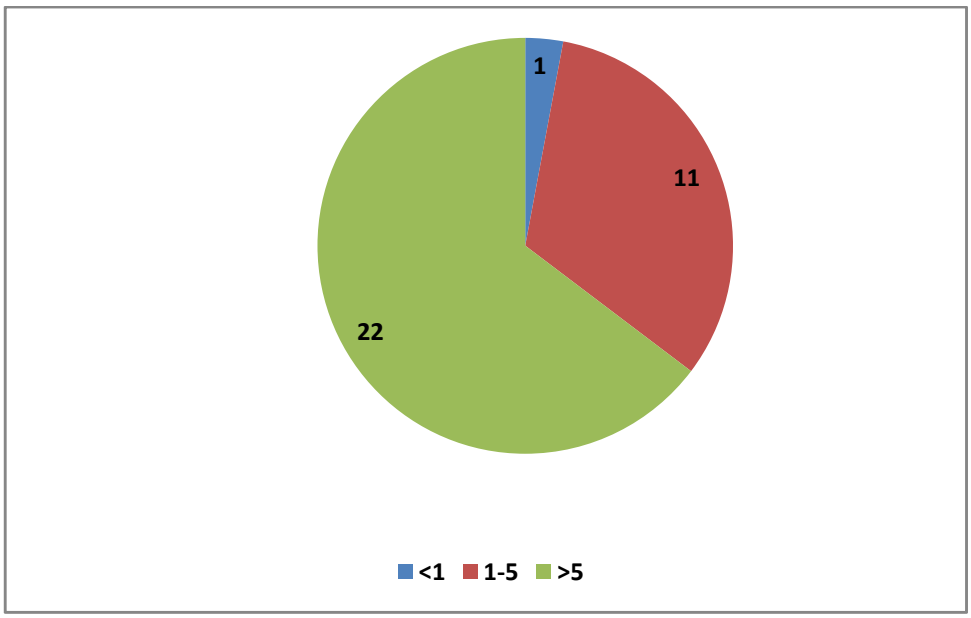


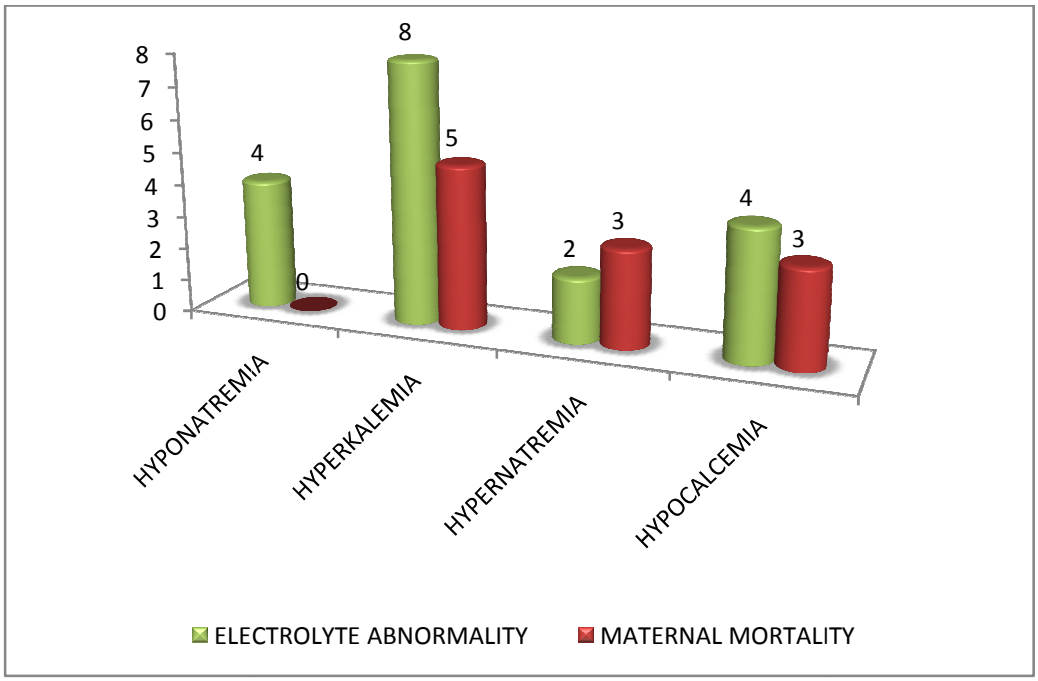
TABLE 16

ELECTROLYTE ABNORMALITIES

ELECTROLYTE ABNORMALITY	FREQUENCY	MATERNAL MORTALITY
HYPONATREMIA	4	0
HYPERKALEMIA	8	5
HYPERNATREMIA	2	3
HYPOCALCEMIA	4	3

CHART 16

ELECTROLYTE ABNORMALITIES



Hyperkalemia, hypocalcemia and hypernatremia carries very bad prognosis

TABLE 17

LEVEL OF URIC ACID IN RELATION TO ACUTE KIDNEY INJURY

URIC ACID	FREQUENCY	MATERNAL MORTALITY
<6	6	3
6-8	13	4
8-10	8	2
>10	7	4

CHART 17

LEVEL OF URIC ACID IN RELATION TO ACUTE KIDNEY INJURY

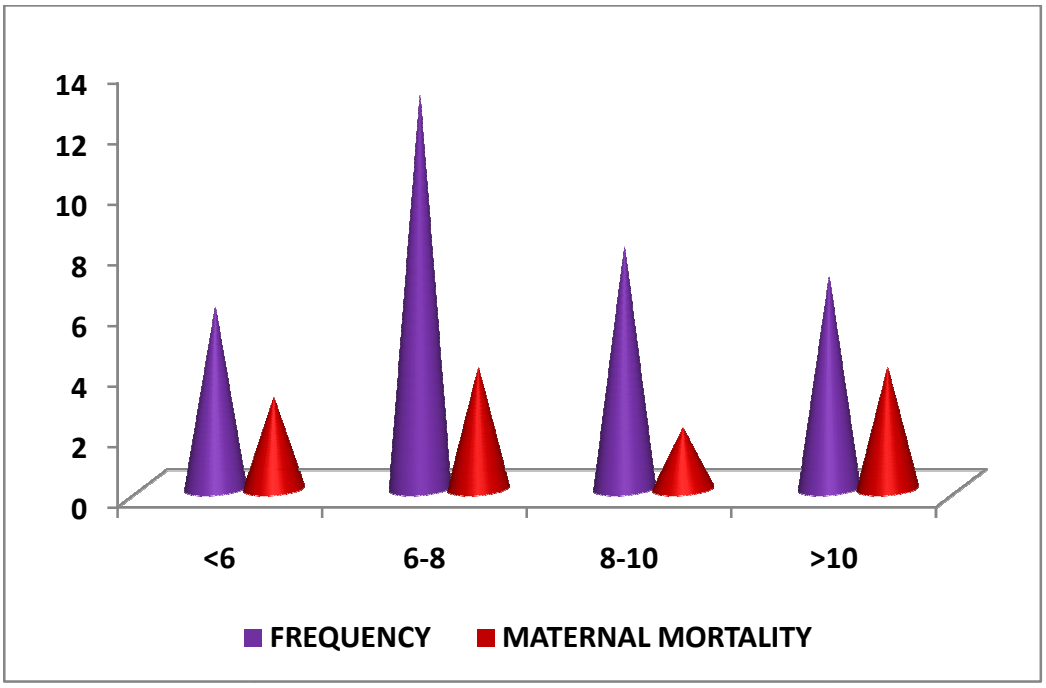


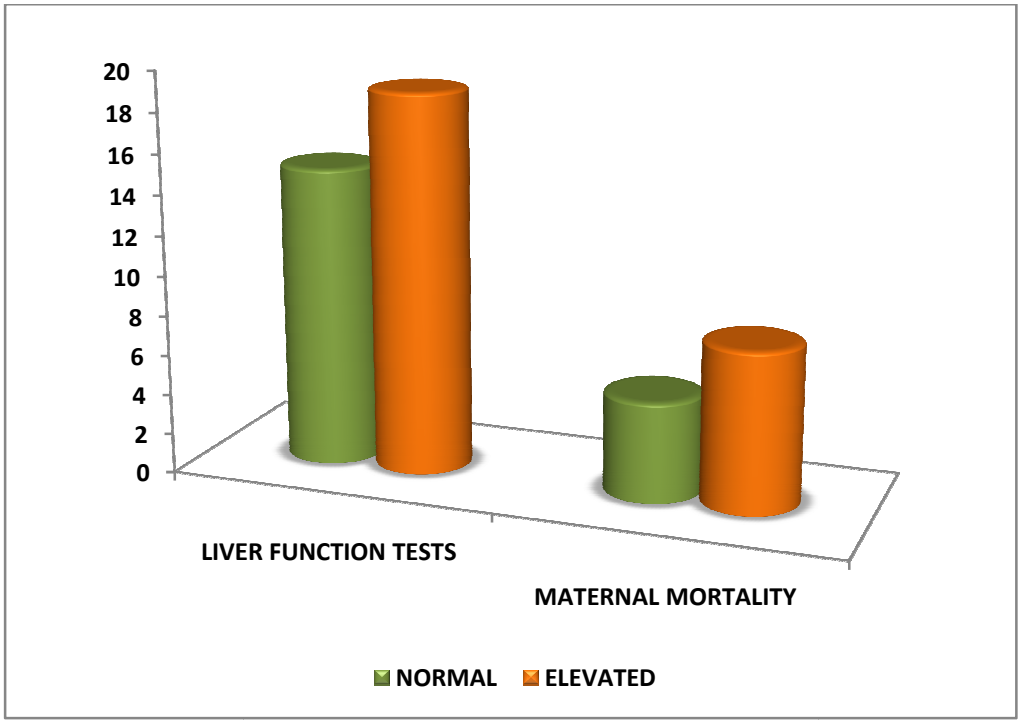
TABLE 18

LIVER FUNCTION TESTS IN ACUTE KIDNEY INJURY

	LIVER FUNCTION TESTS	MATERNAL MORTALITY
NORMAL	15	5
ELEVATED	19	8

CHART 18

LIVER FUNCTION TESTS IN ACUTE KIDNEY INJURY



Abnormal LFT carries bad prognosis.

TABLE 19

MATERNAL OUTCOME

OUTCOME	FREQUENCY	TREATMENT	TOTAL
RECOVERED	21	SUPPORTIVE MEASURES	11
		DIALYSIS	10
DEATH	13	DIALYSIS DONE	7
		NOT DONE DUE TO HEMODYNAMIC INSTABILITY	6

TABLE 19

MATERNAL OUTCOME

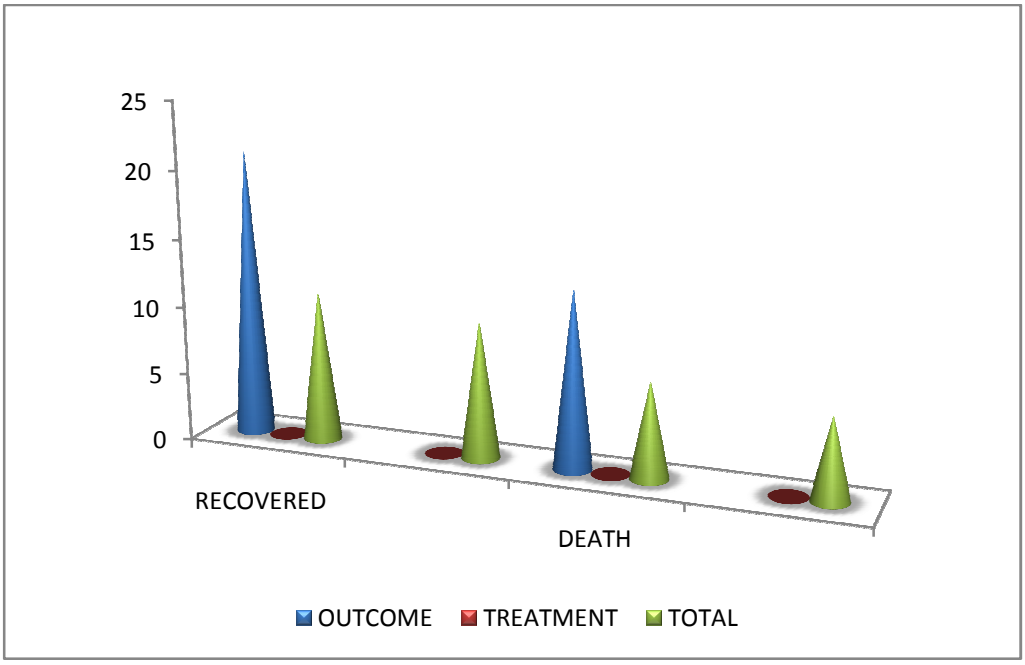


TABLE 20

NEED OF HYPERTENSIVES

HYPERTENSIVES	DRUGS
NO DRUGS	9
HYPERTENSIVES	
<4 WEEKS	16
4-8 WEEKS	4
>8 WEEKS	5

CHART 20

NEED OF HYPERTENSIVES

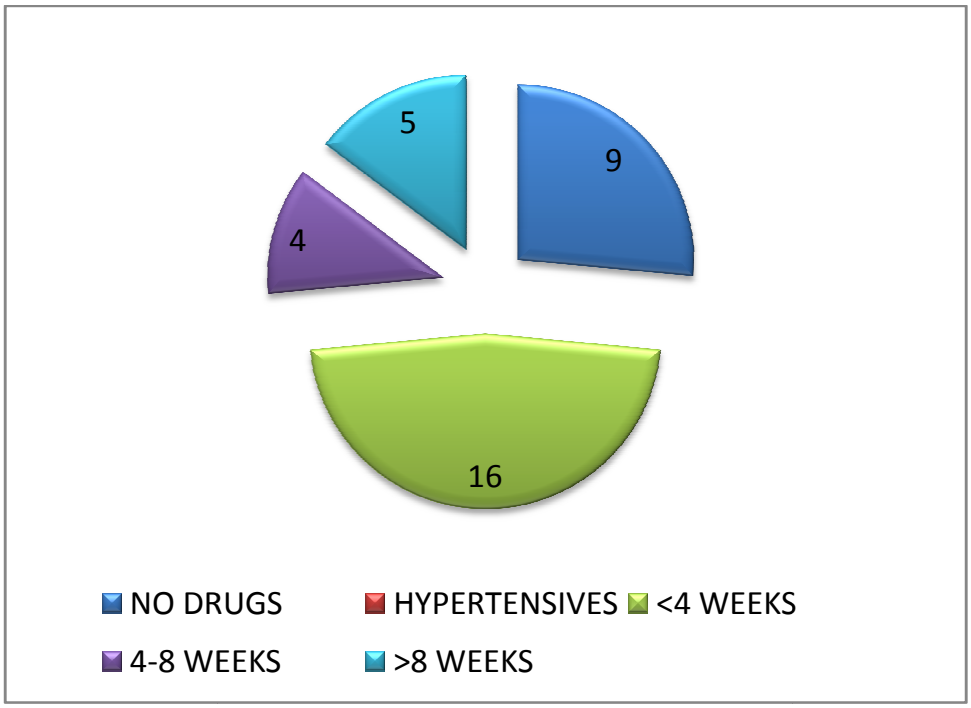


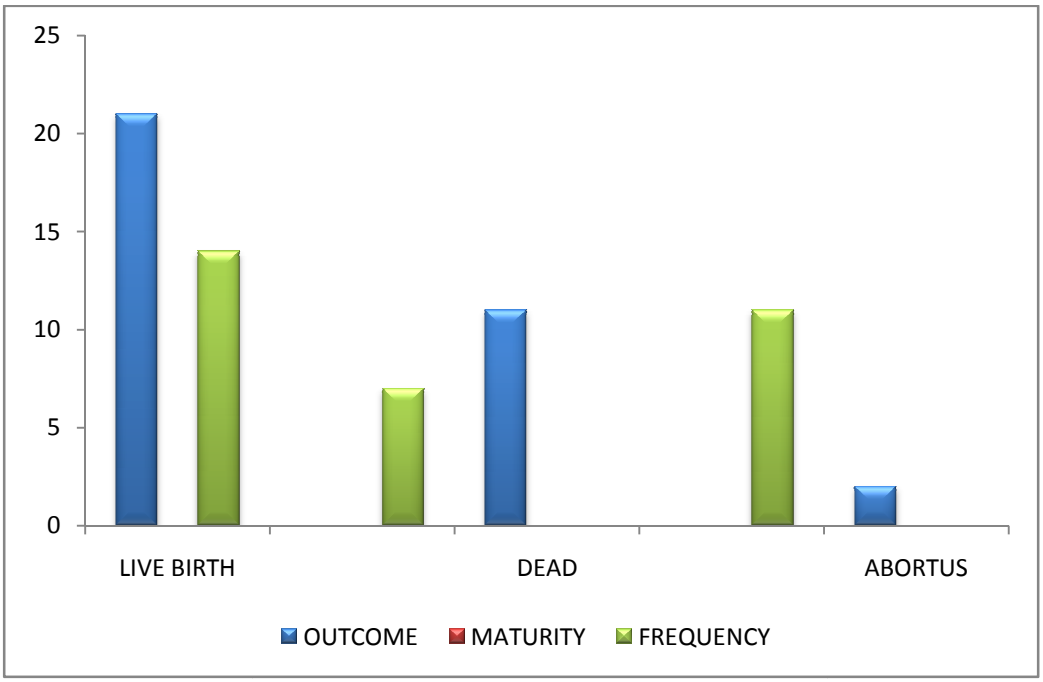
TABLE 21

FETAL OUTCOME

OUTCOME	FREQUENCY	MATURITY	FREQUENCY
LIVE BIRTH	21	TERM	14
		PRETERM	7
DEAD	11	TERM	0
		PRETERM	11
ABORTUS	2		

CHART 21

FETAL OUTCOME



DISCUSSION

The incidence of acute kidney injury among the pregnant women attending Coimbatore Medical College and Hospital, Coimbatore from July 2014 to July 2015 is 4.6 per 1000 deliveries. The incidence in various places by various authors is as follows.

S.NO	AUTHOR	YEAR	INCIDENCE PER 1000 DELIVERIES
1	Namrata khannal	1980	2-3
2	Kilari Sunil Kumar et al (Tripathi)	2006	4.24
3	Mohammed Arrayhani et al (Morroco)	2012	1-2.8
4	Kalki Hymavathi Reddy (Nellore , India)	2015	5
5	Vijay Bhargava et al (Uttar Pradesh , India)	2015	5

Our hospital incidence coincides with the above studies. Our hospital incidence is high when compared to western countries where it is only 0.4% (Williams 2014). This is because of low socio economic status ,reduced awareness of antenatal care and late seeking of treatment.

ETIOLOGY:

In the present study, hypertensive disorders of pregnancy was found to be the most commonest cause accounting for about 58% , the next being post partum haemorrhage about 12% and the third one being anaemia about 9%.

Patients with hypertensive disorders of pregnancy was the major group contributing to AKI in pregnancy. 75% had severe preeclampsia and eclampsia. The study by Mehrabadhi et al and J Prakash showed the incidence to be more in the patients with preeclampsia whereas the study by Archana et al in 2014 showed more incidence in cases of abruption placenta. In 2006 , the study by Kilari et al showed the incidence to be high in sepsis cases.

Because of the wide spread use of antibiotics in septic abortion and legalization of abortion , the incidence has found to be decreased due to sepsis. Nowadays hypertensive disorders of pregnancy remain the major cause for AKI in pregnancy.

AGE GROUP:

In the present study, 47% of individuals were between 21 to 25 years. This correlates with the study of Archana Dambar et al ,2014. This is due to maximum fertility rate of this age,early age of marriage

,illiteracy and low economic status. In developed countries, the age group is between 25- 32 years(Colmont et al)

GRAVIDITY:

In this study, more incidence is seen in multi gravidas. It correlates with the study of Vijay Bhargava et al about 35% among multigravidas.

GESTATIONAL AGE:

44% of the patients presented in the third trimester. This correlates well with the study of Mohammed Arrayhani et al , 2012. In India the study by Ansari et al showed the incidence to be more in post partum period about 75% of the cases.

BIOCHEMICAL PARAMETERS:

In this study, 68% of the patients had proteinuria with serum creatinine levels ranging from 5 to 10 in about 47% of the patients and >11in about 18% of the patients.35% of the patients had an initial urea of about 60 to 100 mg/dl during their presentation. 77% of the maternal mortality is observed in the group with serum creatinine of 1 to 5 mg/ dl. In the study by Mohammed Arrayhani et al mean serum creatinine leading to AKI ranged between 14 to 100 mg /dl.

COMPLICATIONS:

In this study , 12 patients had anaemia. Among them , 5 patients had severe anaemia with hemoglobin <7 g% and rest between 7-10g%. Among the 5 patients, 4 patients died due to PPH. Anaemia and preeclampsia will worsen the prognosis.

MODE OF DELIVERY:

47% of the patients were delivered by LSCS due to obstetric indication and 41% by labour natural. Most of them are preterm delivery. Early delivery by caesarean section is also done in severe cases. However the risk of anaesthesia and the complications due to severity of disease persists and accounts for high morbidity.

FETAL OUTCOME:

About 62% of patients had live births. Among them 14 were term and 7 were preterm. 11 patients had preterm deliveries which were dead born. In the study by Vijay Bhargava et al reported the incidence of about 80% livebirths and 19% dead born.

MATERNAL OUTCOME:

Out of 34 AKI pregnant patients , in this study , 13 patients died . 3 died due to pulmonary edema , 4 due to DIVC (Atonic PPH) and 2 died of ARDS, HUS-2, CVT-2

Of these 13 patients , hemodialysis was done for 7 patients but did not recover from renal failure and died.

Favourable outcome was observed in this study population with the following.

1. BP is maintained below 130/90 mmHg
2. When the biochemical parameters like urea <60mg/dl creatinine <2mg/dl uric acid <7 mg/dl normal LFT. Hb >9 g/dl
3. Blood loss is replaced correctly in time.
4. Anaemia and preeclampsia in combination have a bad prognosis.

In th study by Vijay Bhargava et al, 80 % of the patients recovered completely from the illness whereas 9% died and 9% developed chronic kidney disease.

In the study by Archana dambal , mortality rate was 13%. Other studies have the following mortality rates like Kilari SK et al (24%), Goplani KR et al (18%) Patel et al (15%). In our study , 62% patients recovered from the illness and 38% died . Out of those who survived , 52% recovered completely and 48% developed chronic renal disease requiring multiple hemodialysis.

SUMMARY

A study was made on 34 AKI pregnant individuals who attended Obstetrics and Gynaecology department , Coimbatore Medical college Hospital , Coimbatore to find out the course , morbidity and mortality for a period of one year . The results came as follows.

AKI is common in Antenatal period 65%

Common in the age group 21- 25 years

The complications and incidence of AKI is more common in the third trimester 44%

The most common etiology for AKI is hypertensive disorders of pregnancy about 58%, the second most common being PPH about 12%.

Death occurs most commonly due to DIVC.

29 patients had multiple blood transfusions.

Out of 34 patients , 19 (56%) had oliguria and 4 had anuria (12%). Out of 34 patients, 21 (62%) recovered and 13(38%) died.

Out of 34 patients, 21 had live birth with term deliveries (14) and preterm deliveries (7). Out of 34 patients coagulation profile was done for 5 patients and showed altered profile.

Patients who recovered from renal failure were discharged with a output of 1000 ml per day and serum creatinine level of below 1mg/dl.

CONCLUSION

According to my study, the incidence of Acute Kidney Injury in pregnancy in Coimbatore Medical College Hospital , Coimbatore is 4 – 5 per 1000 deliveries.

Acute Kidney Injury in pregnancy is associated with high maternal morbidity and mortality.

Hypertensive disorders of pregnancy is the commonest cause in 20 cases.

The factors responsible for high maternal mortality in our country are malnutrition, poor access to antenatal care, ignorance and delay in seeking medical advice.

Renal failure developing in the postnatal period carries poor prognosis and has very high mortality rate.

Odema is not a significant symptom.

Patients presented with shock carries high mortality rate.

Patients with anuria had bad prognosis.

Patients presented even late with very high creatinine levels (>5 mg/dl) did not have correlation with outcome.

Patients who had electrolyte disturbances in the form of hyperkalemia, hypocalcemia and hypernatremia had high mortality rates.

Patients with high uric acid levels(10 mg/dl) and abnormal LFT bad prognosis.

Incidence is high in the multi gravidas and in the third trimester.

Early identification of acute kidney injury, early termination of pregnancy , transfusion of blood and blood components , avoidance of nephrotoxic drugs and early initiation of hemodialysis are associated with better outcome.

Perinatal mortality is high due to preterm (< 32 weeks) and its complications.

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ANNEXURES

ANALYSIS OF CAUSES, COURSE AND OUTCOME OF ACUTE RENAL FAILURE IN PREGNANCY

PROFORMA

NAME

AGE

ADDRESS

HOSPITAL

IP NO

DATE OF ADMISSION

DATE OF DELIVERY

DATE OF DISCHARGE

PARITY INDEX

H/O PREVIOUS ABORTION

H/O PREVIOUS IUFD

H/O TREATMENT FOR INFERTILITY

H/O TREATMENT FOR PCOD

H/O MULTIPLE GESTATION

H/O MOLAR PREGNANCY

H/O PREECLAMPSIA

H/O BLOOD TRANSFUSION

H/O FEVER

H/O DIARROHEA

MARITAL STATUS

DIAGNOSIS

MATERNAL COMORBID CONDITIONS

ANAEMIA

CHRONIC HYPERTENSION

RENAL DISEASE

PREGESTATIONAL DIABETES MELLITUS

OBESITY

HYPOTHYROIDISM

RHEUMATIC DISEASE

EXCLUSION CRITERIA

PREVIOUS H/O HYPERTENSION,DIABETES

H/O RENAL DISEASE

USG FINDING OF RENAL SCARRING,SMALL KIDNEYS

ELEVATED CREATININE LEVEL BEFORE PREGNANCY

CLINICAL PRESENTATION

HYPEREMESIS GRAVIDARUM

ABORTION

CONGESTIVE HEART FAILURE

SEPSIS

JAUNDICE

PREECLAMPSIA

ECLAMPSIA

ANAEMIA

HELLP SYNDROME

ANTEPARTUM HAEMORRHAGE

POSTPARTUM HAEMORRHAGE

ACUTE FATTY LIVER OF PREGNANCY

ACUTE TUBULAR NECROSIS

PYELONEPHRITIS

HEMOLYTIC UREMIC SYNDROME

MODE OF DELIVERY

LABOUR NATURAL

LSCS

BABY DETAILS

SEX MALE/FEMALE

ALIVE/DEAD

POSTPARTUM COMPLICATIONS

POSTPARTUM HAEMORRHAGE

ANAEMIA

HELLP SYNDROME

ECLAMPSIA

CVT

CONGESTIVE CARDIAC FAILURE

JAUNDICE

ACUTE RENAL FAILURE

INVESTIGATIONS

	Day1	Day2	Day3	Day4	Day5	Day6	Day
HB							
Platelets							
TC							
DC							
Sugar							
Urea							
Creatinine							
Uric acid							
Urine Analysis							
Sodium							
Potassium							
S.Bilirubin							
SGOT							
SGPT							
SAP							
24 hours Urine Protein							
Intake/ output							
C3							
C4							
ANA							
Urine Spot Protein							
Urine spot Creatinine							
Peripheral Smear Study							
Urine Culture							
Blood Culture							
High Vaginal Swab Culture							
Procalcitonin							

USG ABDOMEN

FUNDUS

CT ABDOMEN

TREATMENT

RENAL FUNCTION SUPPORTIVE MEASURES

DIALYSIS

TREATMENT OF UNDERLYING PATHOLOGY

MATERNAL AND FETAL OUTCOME

CONSENT FORM

I am Dr. K. NEERAJA, carrying out a study on the topic **"A STUDY ON ANALYSIS OF CAUSES, COURSES, AND OUTCOME OF ACUTE RENAL FAILURE IN PREGNANCY".**

My research project is being carried out under the Department of Obstetrics and Gynaecology, Coimbatore Medical College and Hospital.

RESEARCH BEING DONE:

"**A STUDY ON ANALYSIS OF CAUSES , COURSES, AND OUTCOME OF ACUTE RENAL FAILURE IN PREGNANCY**".

SAMPLE SIZE:

34 patients.

STUDY PARTICIPANTS:

Pregnant patients admitted to labour ward in the department of Obstetrics and Gynaecology, Coimbatore Medical College and Hospital, Coimbatore with renal failure.

LOCATION:

CMCH, Coimbatore.

You, Shri./ Smt./ Kum. _____, aged ____ years, S/o / D/o / W/o _____, residing at _____ _____ are requested to be a participant in the research study **titled "A STUDY ON ANALYSIS OF CAUSES , COURSES, AND OUTCOME OF ACUTE RENAL FAILURE IN PREGNANCY".** in Government Medical College Hospital, Coimbatore. You satisfy eligibility criteria as per the inclusion criteria. You can ask any question or seek any clarifications on the study that you may have before agreeing to participate.

DECLINE FROM PARTICIPATION

You are hereby made aware that participation in this study is purely voluntary and honorary and that you have the option and the right to decline from participation in the study.

PRIVACY AND CONFIDENTIALITY

You are hereby assured about your privacy. Privacy of subject will be respected and any information about you or provided by you during the study will be kept strictly confidential.

AUTHORIZATION TO PUBLISH RESULTS

Results of the study may be published for scientific purposes and/or presented to scientific groups, however you will not be identified; neither will your privacy be breached.

STATEMENT OF CONSENT

I, _____, do hereby volunteer and consent to participate in this study being conducted by Dr. K. Neeraja. I have read and understood the consent form / or it has been read and explained to me in my own language. The study has been fully explained to me, and I may ask questions at any time.

Signature / Left Thumb Impression of the Volunteer Date:

Place:

Signature and Name of witness Date:

Place:

Signature of the investigator:

Name of the investigator:

ஒப்புதல் படிவம்

பெயர் :

வயது :

பாலினம் :

முகவரி :

அரசு கோவை மருத்துவக் கல்லூரியில் மகப்பேறு மருத்துவ துறையில் பட்ட மேற்படிப்பு பயிலும் மாணவி **திருமதி. க. நீரஜா** அவர்கள் மேற்கொள்ளும் **கர்ப்ப காலத்தில் ஏற்படும் சிறுநீரக செயலிழப்பு மற்றும் அதன் விளைவுகளும்** பற்றிய ஆய்வில் செய்முறை மற்றும் அனைத்து விளக்கங்களையும் கேட்டுக் கொண்டு எனது சந்தேகங்களை தெரிவுபடுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் முழு சம்மதத்துடனும், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் என்னைப் பற்றிய அனைத்து விபரங்கள் பாதுகாக்கப் படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபணை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் இந்த ஆய்வில் இருந்து நான் விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

இடம்

தேதி

கையொப்பம் , ரேகை

KEY TO MASTER CHART

G	-	Gravidity
P	-	Parity
L	-	Live Birth
A	-	Abortion
AN	-	Antenatal
PN	-	Postnatal
P	-	Present
A	-	Absent
N	-	Normal
LN	-	Labour Natural
LSCS	-	Lower Segment Caesarean Section
PT	-	Preterm
T	-	Term
UW	-	Under Weight
O	-	Obesity

MASTER CHART